PROCEEDINGS OF THE 1956 REGIONAL MEETINGS ON FOREIGN ANIMAL DISEASES

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Stillwater, Oklahoma - March 19-21
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FOREWORD

Material contained in this publication represents summaries of most of the discussions and talks presented at the several regional meetings on foreign animal diseases held by the Agricultural Research Service in cooperation with the State regulatory officials during the spring of 1956. This material is recent in its preparation and can be used to provide information and promote discussion on the potential threats of foreign animal diseases on livestock of this country. Precautions taken by the Federal government to help prevent the entrance of foreign diseases into the country are explained as well as the cooperative State-Federal disease control programs, and the State-Federal emergency disease control and eradication programs. Several serious foreign diseases with symptoms and pathology similar to diseases already present in this country are discussed and the need for promptly reporting unusual and foreign-type diseases is emphasized.

These meetings, together with material contained in these proceedings, will provide a better understanding of the problem associated with foreign disease prevention and control and will help to encourage the cooperation of all segments of the veterinary profession and livestock industry in the protection of livestock and poultry of this country from foreign diseases.

Frank A. Todd

Frank A. Todd
Assistant to the Administrator
Agricultural Research Service

GREETINGS

Theodore S. Gold
Assistant to the Under Secretary
U. S. Department of Agriculture
Washington, D. C.

I am more than happy to bring to all of you--residents of Connecticut, as well as others--the greetings of the Office of the Secretary of the United States Department of Agriculture. We feel that this meeting is an important one, and that you are considering important questions. We are pleased that you have been willing to gather here for this conference, and I am pleased to participate with you in these discussions.

Under Public Law 920, the Administrator of the Federal Civil Defense Administration is granted very broad powers for certain emergency operations. He is charged, however, with utilizing the facilities and "know-how" of other Federal agencies wherever possible. He is prohibited from establishing duplicate services unnecessarily. Acting under these conditions, the Administrator of the Federal Civil Defense Administration has delegated certain important responsibilities to the Secretary of Agriculture.

They are: (1) The formulation of national food plans, looking toward adequate emergency food supplies for attacked and support areas; (2) the suppression of fires in rural areas caused by enemy action; (3) protection of crops and livestock against subversive attack through biological or chemical warfare. This last item, of course, is the primary reason for this meeting.

Within the Department, the Secretary has re-delegated these responsibilities to those Services which are best equipped for them by technical know-how and related routine programs. The food planning problem is delegated to the Agricultural Marketing Service and the Commodity Stabilization Service. The fire control program is delegated primarily to the Forest Service with an assist from the Extension Service. The protection of crops and livestock against biological and chemical warfare is delegated to the Agricultural Research Service. Secretary Benson has seen fit to name me to coordinate all the Civil Defense activities of the Department of Agriculture, which is the reason for my being here.

Now, I don't want to be an alarmist, and I do not want my remarks to be misinterpreted, but I do want, in all seriousness, to impress upon you folks that I feel this meeting is important. It is, perhaps, significant that this past winter at some sort of top-level Soviet meeting, it is reported that their military leaders indicated they now have the ability and know-how to conduct biological warfare.

I think it is fortunate that the normal work of persons like yourselves equips you to cope with these unforeseeable emergencies or catastrophes. The better our preparations against biological and chemical warfare, the greater our capability to protect our crops and livestock from natural outbreaks of disease and pests. The healthier our crops and animals, and the stronger our organization to prevent natural spread of pests and diseases, the greater our capability to resist subversive or overt warfare through these media, if it should ever come.

I do think that, without hysteria, and without panic, we should plan seriously for our defenses. This is a real threat, and we could be losing a war, or even losing our cherished American freedom and way of life, if we neglect our responsibilities in these areas.

I want to commend all of you for your serious, intelligent, hard-working approach to these subjects. I feel that this meeting is well worth-while, although I am sorry that there are not more actual practitioners here. The practitioner is the man on the firing line, and our first line of defense. I hope that you folks can take homewith you, and get to the practitioners in your states, the information which you are receiving here.

Again, thank you for being here, and thank you for letting me meet with you.

INTRODUCTION TO REGIONAL MEETINGS ON FOREIGN ANIMAL DISEASES

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It is a pleasure to have so many of you here to participate in a discussion on foreign animal disease problems. As you may know, regional meetings on foreign animal diseases were held last spring, attended primarily by State and Federal regulatory officials. The purpose of those meetings last spring was to present to the regulatory officials (1) the latest information we had in regard to a number of these diseases, (2) how they may be introduced into this country, and (3) to discuss with these officials how the State and Federal governments can best cooperate in an emergency disease eradication organization for the rapid extirpation of any diseases which may gain entrance into this country.

The purpose of this year's meetings is to familiarize veterinarians who will most probably be the first to investigate unusual occurrences of diseases with methods of differentiating between certain endemic and exotic diseases. We have invited from each State the practitioners, the directors of the State diagnostic laboratories, the veterinarians of the veterinary departments of agricultural colleges, extension veterinarians, representatives from veterinary colleges, including the clinicians and certain instructors, as well as members of the senior classes, and veterinarians from commercial diagnostic laboratories. We also have with us the veterinarians who have been selected to head up the emergency disease eradication program in each State in this area. Since several of the diseases to be discussed are transmissible from animals to man and would provide a public health problem, we have also invited the State public health epidemiologist and the director of public health laboratories.

Since the beginning of World War II, there has been a great deal of discussion of biological warfare being directed against the animals of this country. Aside from its economic effect, there are good reasons to believe that an enemy might attack this country in that way, since many of these diseases are also communicable to man. Such an attack would not only have the result of diminishing our food supply, but would also affect the health of the livestock producers whose production capabilities would be taxed to the utmost during times of war. Certainly, we have learned that our country does not have to be engaged in an all-out war for us to have problems presented relative to the control and eradication of animal diseases. Therefore, the plans we are making are not only to meet attacks of biological warfare but are just as applicable to cope with the accidental introduction of foreign diseases or unusual occurrences of endemic diseases.

Scrapie and bluetongue are two examples of diseases which have gained entry into this country, apparently through an accidental route. The spread of vesicular exanthema to 42 States and the District of Columbia once it had left the confines of California is ample evidence as to how fast these infectious, communicable diseases may be disseminated once they gain entrance to our channels of trade.

Our full-time State and Federal forces are entirely too few to depend upon for the early detection of foreign or unusual manifestations of endemic diseases. We know we are almost entirely dependent upon the practitioners, workers in diagnostic laboratories, extension veterinarians, college clinicians and research workers. We are gathering you together to tell you what we know, what we're trying to do to keep them out and what preparations are being made to be prepared to handle outbreaks if they should occur. Past experience indicates that they will occur from time to time.

If we are to cope successfully with these problems, we must have available information pertaining to these diseases. We must be able to obtain a correct diagnosis, if such a disease should occur, and must learn how it is being transmitted, its hosts, its effects, and its extent. All of this information would have to be gained through epizootiological investigations, and such investigations would require personnel experienced in such procedures. Then we, the State and Federal agencies, must have a plan and an organization for dealing with them.

Attention has been given in connection with foreign animal diseases to laboratory diagnostic work. Late in 1953 a group of outstanding poultry pathologists met in Washington to discuss how foreign poultry diseases could best be prevented, detected, and eradicated. Since poultry diseases are principally diagnosed in the laboratory rather than in the field, it was recommended that a survey be made of all poultry diagnostic laboratories in the United States and that regional coordinators be designated for coordinating the diagnostic facilities of the several areas of the United States. At the second meeting of the collaborators in June 1955 it was recommended that the diseases of poultry be placed on a comparable basis with diseases of livestock, and to accomplish this, that a section on poultry diseases be established within the appropriate branch of the Agricultural Research Service. At this same meeting the recommendation was made that the central laboratory in each area should proceed to establish within the region an adequate pathological reference museum (both gross and microscopic), as well as a library consisting of subject matter not readily available, Kodachrome slides, charts, and other visual aids.

The work with regard to these reference laboratories has not proceeded as rapidly as it might have, due to the fact that the Animal Disease

Eradication Branch has had no section on poultry diseases, and therefore no one to establish these laboratories. However, a section is now being established, and the section chief will devote much of his time to this phase of the work.

Since it was deemed advisable to close down the laboratories in Washington last summer, space for diagnostic work has become more critical than ever. We have asked the regional collaborators to get together here to discuss the establishment of reference laboratories for all animal diseases.

We are here to discuss all these problems with you, and by reviewing what we know about these diseases, to bring everyone up-to-date as to what has been done and what is planned for the future, and to solicit your complete cooperation.

In summary, therefore, I should like to emphasize the following points:

I. <u>Purpose of Meeting</u>

1. Bring veterinarians from various fields together to participate in discussions on the challenge that lies before all of us: "What to do about foreign disease should it be found in this country?"

2. Present discussion on the foreign diseases that appear to be the most dangerous to the livestock industry of this country if they should gain entrance into the country.

3. Present highly qualified individuals who have had actual experience with these diseases to discuss them in detail.

4. Cite ways in which diseases can be introduced into the country.

5. Emphasize in detail the similarity of symptoms in diseases presently encountered and those observed in foreign diseases.

6. Emphasize the possibility of one or more of these diseases becoming well established by being diagnosed as one of our endemic diseases.

7. Need to recognize these foreign diseases early and report them to regulatory officials.

8. Discuss the plan to control and eradicate these diseases.

II. <u>Objectives of Meeting</u>

1. Acquaint you with the problems encountered in the control and eradication of foreign diseases.

2. Seek your cooperation in the diagnosis and early reporting of any condition that may resemble these foreign diseases.

3. Acquaint you with the plan that is being developed to combat these diseases. Importance of an active State-Federal organization for emergency disease control that can act immediately. Steps to be taken if unusual conditions appear.

4. Acquaint you with the part that we are expecting you to play in the defense of the livestock of the country.

5. Acquaint you with what the Agricultural Research Service is doing to try to prevent the entrance of these diseases, what related research is being conducted and what diagnostic services are available.

FOREIGN ANIMAL DISEASES - A THREAT TO OUR LIVESTOCK

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The presence or absence of animal diseases is a major determining factor on the health and welfare of any nation. The presence of disease results in loss of livestock resources. It determines the economic availability of such products. The lack of proteins, fats, and other byproducts obtained by the healthy, abundant livestock can regulate the health and welfare of the country. Not only is the control of existing animal diseases important in helping to provide adequate products of animal origin, but also the prevention of the introduction of new and additional diseases into a country or community.

There are many diseases affecting all species of animals and birds found in various parts of the world. These diseases can vary from those that produce only slight debilitation to their infected host to those that carry with them an extremely high mortality. Some may affect man. Many of these diseases are endemic in various areas of the world and some appear sporadically. They are all capable of being transmitted from one part of the world to another.

In man's efforts to produce healthy livestock and an adequate food supply, he attempts to protect his animals from disease. Nations in establishing defenses against the introduction of the more devastating diseases have developed or created animal populations that are susceptible to those diseases that have been restrained.

The countries of North America now enjoy freedom from such dangerous and devastating epizootic diseases of animals as foot-and-mouth disease, contagious bovine pleuropneumonia, rinderpest, Rift Valley fever, fowl plague, fatal form of Newcastle disease, and several serious diseases of swine. Upon several occasions, these countries have been successful in ridding themselves of foot-and-mouth disease. In the past, these diseases have periodically ravaged the various animal populations of the world, at times causing almost complete destruction of herds and flocks. In those countries, particularly in the Orient, that have failed to effectively control the more dangerous animal diseases, a poor state of livestock and agricultural industries exists. This is a major factor contributing to the serious human food shortages.

It is recognized that the establishment of adequate control measures for livestock diseases does lead to a considerable increase of supplies of foods of animal origin, as well as byproducts. In those areas where

modern science is utilized for the control of the more devastating diseases of livestock, marked results have been obtained. There are major differences, however, between countries, in the methods that are used to prevent and control animal diseases and in the general standards of the application of such measures.

Preventive medicine has developed rapidly during the last decade in scope, volume, and in effectiveness. This progress is due to the increased knowledge gained through important discoveries of bacteriology, biology, chemical and sanitary sciences with new and fresh observations resulting from epidemiological, epizootiological, and ecological studies.

We are becoming more aware of the fact that most disease agents may affect or may reside in several host species and that this may be accomplished by the organism selectively adapting itself to new hosts. This emphasizes the complicated problems associated with animal disease control and preventive medicine. These facts should also remind us that the approaches to disease control and preventive medicine are very seldom accomplished on a single species basis and that these problems cannot be immediately nor arbitrarily segregated into areas of human or veterinary medicine.

In reporting that a particular disease affects only one species of animal or that a disease of animals is non-effective to man, we are only reporting on our observations up to the present time -- we are dealing with a dynamic rather than a static situation with the possibility of fresh discoveries in the future.

In the past, we have looked upon a number of these animal diseases as problems that were peculiar to Asia, Africa, Europe, or wherever they happened to be. With the advent of planes and the build-up of international trade and travel, today foreign animal diseases have taken on a new and added importance.

Even though rigid and very effective precautions are continually in operation to help prevent the introduction of foreign diseases into this country, there still remains and always will remain the possibility that some diseases may gain entrance.

In quickly reviewing past outbreaks of unusual or foreign diseases, we have found that they have been introduced and spread by a number of means, including the feeding of raw garbage, importations of contaminated animal feeds, the careless handling and disposal of cultures of agents that have been used experimentally in the country, contaminated biologics, smuggled livestock and birds, insect vectors, perhaps even migratory birds, and animals imported as healthy carriers or with the disease having an unusually long incubation period. It might even be

possible that the recent hurricanes we have experienced during the past several years may be a factor in the introduction and spread of disease.

In the past we have enjoyed a time factor in the importation of livestock into this country. Shipments of livestock by sea from Europe, Asia, or Africa took from 15 to 40 days. This period of time provided an opportunity for most diseases to develop signs that could be observed, and proper control measures carried out prior to or at the time of arrival.

Consider modes of travel. This is the air age. International travel and freightage are at a peak. A lot of this is by air. Even our livestock - 76 percent of the poultry, 54 percent of the horses, and 1 out of 9 sheep, goats, hogs, and zoo animals that are imported - comes in by air. Ship dockings have doubled and international air flights have increased 17-fold since prewar. One-third of those flights harbor plant material that must be barred.

Livestock's travel may be quicker than the incubation of its diseases - too quick for symptoms of diseases to show up.

International airports miles inland from the coast are a new inner border. We must redeploy and man that line, too.

Our inter-continental tourists of today also provide a potential hazard that is of concern to this problem. Many people upon entering or returning to this country bring gifts of great quantities of dried sausage, salamis, and other meat delicacies in the baggage which Customs seizes for disposal. As much as 160,000 pounds of meat have been seized from passenger baggage in one year. From 50,000 to 75,000 pounds of restricted meat is seized each year from passenger baggage. Since Customs inspection is not carried out on all incoming baggage, there may be quantities of these products being brought in. These items are potentially dangerous because most of these sausages are made from ground beef, pork and veal - seasoned and dried but not cooked. The danger lies in the fact that a lot of our people might care little for such products and when their friends or relatives have gone they may throw it into the garbage can and from there it might be fed to livestock.

Foreign animal diseases that might gain entrance into this country either by accident or by deliberate acts of sabotage can quickly become widespread among the livestock and poultry unless we are prepared to recognize them promptly and immediately take measures to combat their introduction and apply sound control measures. Some may be confused with diseases already present in this country, a differential diagnosis being required in almost each instance.

The outbreak of foot-and-mouth disease in Canada several years ago was a classic example of what might occur in this country. In this case, the disease appeared in a most unexpected location -- in a very remote locality of a central Canadian province -- far from a border point where the disease could have gained entrance. The disease was vesicular in nature and because of its location, it was tentatively diagnosed as vesicular stomatitis. Three months passed by before it was finally confirmed as foot-and-mouth disease. In the meantime, it, of course, spread. This could happen in our own country. There are other serious foreign animal diseases that could cause the same problems if they made their appearance in this country. We must constantly keep this in mind.

One of the principal problems in dealing with foreign animal diseases is the lack of readily available information pertaining to these diseases, together with the lack of experience in handling them; thus, we must become better acquainted with foreign diseases and be in a better position to recognize them if they should gain entrance into the country. The prompt recognition and the prompt reporting of the disease, of course, is a primary requirement for successful control and eradication. The sooner the disease is recognized and reported, the quicker action can be taken against it. The speed with which the action is taken will determine, to a great extent, whether the disease can be initially eradicated or whether a slower and more costly procedure of controlling it with the use of biologics and other measures will have to be used.

Another problem in dealing with foreign animal diseases is diagnosis and confirmation. Many times a differential diagnosis will be necessary because for almost every foreign animal disease that threatens the livestock of this country, there is one or more disease already present in this country that presents symptoms that are similar or in some cases identical to them. We must continually keep this problem in mind. The presence or the suspected presence of a disease that may be thought of as a native disease may in reality be masking a more serious foreign plague.

The feeding of raw garbage containing meat scraps from animals infected with various diseases has been found through experience to be a means of perpetuating, introducing and spreading certain contagions.

At least two outbreaks of foot-and-mouth disease in the United States were associated with the feeding of raw garbage. The very sudden and unexpected nationwide spread of vesicular exanthema emphasized the feeding of raw pork scraps to swine as a means of spreading disease.

During World War II, the appearance of hog cholera in countries throughout the world that had not encountered this disease before were related

to the feeding of pork scraps from the U. S. Army' messes in which frozen pork products were used in the menus of American and allied messes.

Contaminated animal feeds have also been associated with the introduction and spread of animal diseases. In 1952, Finland traced an outbreak of foot-and-mouth disease to feeds which had been imported into their country from Russia.

An unusual outbreak of anthrax in the United States at about the same time was found to be caused by a shipment containing contaminated bone meal imported into the United States and used as a supplement mineral in swine feeds.

Contaminated biologics have been incriminated in several instances as a factor in introducing and spreading animal diseases. An outbreak of foot-and-mouth disease in the United States followed the importation of a culture of smallpox vaccine from Japan.

Gregg of England describes an experience in Scotland where scrapie appeared in several flocks of sheep following the use of louping-ill vaccine. Investigations revealed that sheep used to prepare a particular lot of the vaccine in question were the progeny of scrapie infected ewes.

Outbreaks of Newcastle disease have followed the use of the biologic intended to prevent the disease. On one occasion in the Midwest, a widespread outbreak of pullorum disease followed the use of Newcastle disease vaccine and it was found in flocks that had been pullorum free for a number of years. Investigations in this case showed that the eggs used in the preparation of the Newcastle vaccine had originated in pullorum diseased flocks.

The careless disposal of a laboratory culture of fowl plague virus resulted in the appearance of this disease in several flocks of chickens in the eastern part of the United States about 1924. The virus was brought into the United States illegally by a laboratory worker the previous year.

All through history the smuggling and the illegal movement of livestock has caused the introduction and spread of various diseases of livestock and poultry. The smuggling of psittacine-type birds into the United States from foreign countries has resulted in many human cases of this disease and the widespread incidence of the disease in this industry.

The smuggling of both cattle and swine between various countries and areas of Germany immediately following the war resulted in the

appearance of foot-and-mouth disease. The uncontrolled movement of horses by the German army during and following the war introduced glanders, dourine, and infectious equine anemia into countries which had been free of the disease for a number of years.

The importation of animals during an incubation period and especially those diseases with an unusually long incubation period have been the means of introducing certain diseases into countries. The epizootiological investigation of an outbreak of scrapie in the United States and Canada indicates that this disease with a possible incubation period of from several months to several years is not prevented by the established quarantine practices regulating the importation of livestock.

Another problem related to importation of livestock is the presence of inapparent or healthy carriers. Under these conditions, the host animal may carry the infective agent but does not show symptoms or signs of the disease. Reports on the capability of cattle carrying the virus of bluetongue without showing symptoms is an example.

A classic example is the disease African swine fever which can be carried in the blood of a wild hog, the wart hog, and the bush pig without the slightest indication that the virus is present in the blood. When these inapparent carriers come into contact with a susceptible host the disease is then transmitted. As epizootiological investigations are conducted on animal diseases, the problem of inapparent carriers in both domestic and wildlife becomes more evident.

Insect and arthropod vectors too can play an extremely important part in the introduction and dissemination of disease. The early epidemiological investigations of Venezuelan encephalomyelitis revealed that the disease was transmitted from the mainland of Venezuela over to the island of Trinidad by the culicine mosquito, *Aedes taeniorhynchus*, a vector capable of flying the six-mile stretch of water separating the island from the mainland. The insect *Mansonia tittilans* has also been incriminated as the vector responsible for distribution of the disease on the island.

Increased and rapid traffic in livestock within a country adds to the problems of disease prevention and control. In the United States, it is getting to be common for a steer to have lived in two or three states before going to the packing plant for slaughter. Livestock mingle in flight and they come from great distances. The marketing practices of the United States increase many fold the chances of disease spread.

When hog cholera was first seen in Ohio in 1833, it required almost 30 years for the disease to appear in some states. The cattle fever

tick moved at the rate of four miles a year during the 1870's. In contrast to that, when vesicular exanthema broke out of California in 1952, the disease was diagnosed in 16 states within a two-week period.

Migratory birds have been incriminated in a number of cases as a means of introducing and spreading disease. Outbreaks of foot-and-mouth disease in England have been associated with the flight or the presence of birds from the European continent. The fact that birds are considered a reservoir for the encephalidities makes them an ever present potential source of this infection so that migratory birds may be looked upon as another means for the introduction and spread of disease.

More recently the hurricanes that have appeared in ever increasing numbers have been followed by the increase in insect populations and the spread of certain insect diseases of plants from areas in which the condition had been confined for several years. The gypsy moth which had been confined to the New England states for a number of years by means of natural barriers such as mountains was found beyond the quarantine areas following a hurricane. Salt water mosquitoes have increased in great numbers along the eastern shore of the United States following the hurricanes of 1955.

We should not forget our experience with Newcastle disease when the first cases made their appearance in the United States. Appearing in an apparently atypical form with a relatively low mortality as compared to the fatal forms which were experienced in Asia, Africa, and parts of Europe, it was not recognized. Under these conditions, adequate control measures were not carried out and the disease was allowed to spread throughout the country before it was finally recognized. As a result of this experience, this disease has now become established within the poultry industry of the United States.

A similar experience was associated with bluetongue in sheep where it had been mistaken for several diseases of sheep that do produce symptoms and lesions that are similar to bluetongue. Here again the mortality of the strain appearing in the United States was extremely low as compared to the highly fatal strains reported from South Africa.

These facts are important and must be continually kept in mind as we see and work with new or unusual diseases that do appear from time to time in our livestock.

Diseases that are transmitted by arthropod vectors present a peculiar problem. In many of these diseases by the time the disease has been diagnosed the agent will have been widely spread and established in the vector host. Under these conditions, control or eradication procedures are extremely difficult and complicated. Certainly under such

conditions the slaughter of infected animals without adequate controls and eradication of the vector will give little protection against the spread of the disease. There are several diseases looked upon as being foreign to this country at this time that are transmitted by arthropod vectors. In several cases, the vector is already present within the United States and requires only the presence of the agent.

Bacteriological warfare, if it comes in the hot war sense, will be nothing new. It is here and has been here in the every day crisis, the routine emergency, confronting those who, day in and day out, year in and year out, battle the diseases and insect pests of plants and animals. Peacetime warfare against pest and disease has been the basic training of men engaged in this line of work. A larger emergency can only put the program into high gear, mobilize the reserves, throw shock-troops into the fight where the battle is fiercest. Things will have to be learned, some of them fast and some of them the hard way. But bone-hard work and high ingenuity on the part of dedicated Federal, State, and private workers will recognize the problems and map the strategy. They must know where to go and what to do in a national emergency.

WHAT PRECAUTIONS ARE TAKEN TO PREVENT INTRODUCTION OF FOREIGN DISEASES

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In the days before the advent of the airplane and the fast-moving ship, the introduction of disease by animals or products was not of quite the same moment as it is today. Prior to 1850 the movement of livestock was confined to those animals which would accompany the pioneer as he moved across the country and spaces were so vast that the possibility of the spread of infection was slight. In order to give you an insight as to how disease was introduced into the United States, I would like to tell a story which actually happened.

About 100 years ago, a British vessel with the name of George Washington docked in the port of New York, almost opposite the window of our office at 45 Broadway. On this ship were some cattle which had been brought along to furnish milk and food for the crew members and passengers. The particular cow that furnished the milk had gone dry so that the master of the vessel proceeded to take the cow off the ship to a rural district then known as "The Bowery", where he traded this cow for a fresh cow. This transaction being done, he returned to the ship and sailed away. The cow he traded, however, was infected with contagious pleuropneumonia which spread over the United States, particularly the eastern seaboard and as far west as Ohio, and for a number of years this particular disease was the scourge of the infant livestock industry in the United States.

Luckily, even in those days, there were people farsighted enough to consider the eradication of contagious pleuropneumonia of grave importance and, in about six or seven years after the program was started, this disease was eradicated from the United States. The introduction of this particular disease into the United States in this manner is a situation which could not occur under our present laws and regulations.

In 1884 the Congress established what was formerly known as the Bureau of Animal Industry and provided for certain quarantine stations to be established at the several ports of entry to guard against just such an occurrence. Since that time, our work has taken on considerable more activity in that we are not alone concerned with the introduction of live animals from known foot-and-mouth disease or rinderpest infected countries, but also with the possibility of the introduction of such diseases through importations.

You have heard of the danger of foot-and-mouth, rinderpest, and these other exotic diseases and the grave situation this country is placed in, in trying to keep these diseases out -- what do we do about it?

First in importance, I would class ships' garbage, and meat from passenger baggage. There have been two outbreaks of foot-and-mouth disease directly traceable to garbage from a ship.

Since the law, Section 306(a) of the Tariff Act of 1930, has been in force, we have controlled the movement of garbage, meats and animals from ships in the following manner:

All ships entering our territorial waters at Public Health quarantine must declare whether they are carrying ships' stores of meats from foot-and-mouth disease or rinderpest countries; or ruminants or swine from such infected countries. In case animals are aboard, Animal Inspection and Quarantine is immediately notified and drastic action is taken to see that these animals are slaughtered and disposed of in such manner as to eliminate any danger of exposure to our livestock.

No garbage from ships carrying foreign meats may be brought off a ship unless it is in a tight container and brought off for incineration under the supervision of an Animal Inspection and Quarantine inspector.

Hides and skins from infected countries are allowed entry into this country only if the skins are hard-dried, having the appearance of parchment, or pickled in mineral acid, or if they are brought into the country for restricted handling in establishments approved for the handling of restricted products where the product is thoroughly disinfected by the use of a sodium bifluoride disinfectant for a 24-hour period.

Wool is allowed entry if free from blood stain and reasonably free from animal filth or dirt.

Bristles are allowed entry if cleaned, washed and sorted before coming to the United States.

Fresh, chilled, or frozen beef, veal, lamb, and pork are prohibited entry into the United States from infected countries. Cured or cooked meats of these classes may be admitted to this country if thoroughly cured or cooked and brought into the country to plants where it is to be processed before being sold to the trade.

Hay and straw packing material is prohibited from infected countries unless such hay and straw is disinfected in such a manner as to destroy the rinderpest and foot-and-mouth disease virus. This may be by use of live steam and formaldehyde.

Animal glands are the only fresh products allowed entry from a foot-and-mouth disease or rinderpest infected country, these to be brought in and processed for pharmaceutical purposes only. A great deal of care must be used in the handling of such products. They are sent to approved establishments where the entire process of manufacture is adequate to guarantee a disease-free final product.

Animal stomachs and rennets are allowed entry if dried to the consistency of parchment, or they may be entered as restricted products in establishments where the product is handled in such manner as to prevent the introduction of foot-and-mouth disease and rinderpest.

Bone meal, hoof and horn meal, meat meal and tankage. Bone meal for feed or fertilizer must be a steamed or degelatinized product with a nitrogen content of 2 percent or less. Hoof and horn meal may be imported if it has been heated to a degree of heat to guarantee freedom from foot-and-mouth disease, rinderpest, and anthrax. Tankage and meat scrap may be imported if accompanied by a certificate showing it has been heated to a temperature to destroy foot-and-mouth disease and rinderpest virus, and to contain 35 percent bone phosphate of lime, or less.

Domestic cattle, sheep, and swine are prohibited entry into the United States from any country where foot-and-mouth disease or rinderpest exists.

All classes of animals, ruminants, swine, and poultry, except horse stock, can come to the United States (except from Mexico and Canada) only after a prior permit has been secured for such entry. When a permit is issued, certain requirements are set up, such as 60 days at the port of export, veterinary certification by government veterinarians of the country of origin, including certain diagnostic tests. After these requirements have been met, the animals are allowed entry into our ports. Before being unloaded from the plane or ship, they are given a complete veterinary inspection by our port veterinarian and, if everything is in order, allowed to move forward to quarantine and such tests as are required there.

Horses are inspected on the carrier on arrival and if they come from certain countries are held in quarantine for a blood test for trypanosomiasis and glanders.

Wild ruminants may be permitted entry provided they are destined to an approved zoo where they can be held in strict control and not be moved about the country.

Since the recognition of African swine fever in Africa, wart hogs and bush pigs for zoological parks have not been permitted entry.

Biologics, vaccines, sera, and various hormone and glandular substances all are suspect from rinderpest or foot-and-mouth disease countries and are held under control until thoroughly investigated as to whether it is safe to permit their entry.

In this day and age of seeking additional knowledge, we are constantly requested to allow the entry of disease cultures, vectors, and blood samples for educational institutions and laboratories. Each product is thoroughly investigated and its merits considered before it is permitted entry.

Semen from cattle, sheep and swine from foot-and-mouth disease or rinderpest countries is prohibited.

It will be interesting for you to learn that since the end of World War II there has been a great movement of people between the United States and Europe -- people who have been going back to the homeland, or people who have been coming to visit relatives here, and it would seem for a while that almost everyone in foreign countries thought that we here in America were hungry for meat and for that reason they brought great quantities of dried sausage, salamis, and other meat delicacies in their baggage, which Customs seized for us for disposal. We have in the past seized as much as 160,000 pounds of meat out of passenger baggage in one year, and currently we are taking from five to six thousand pounds a month. Customs inspection of passenger baggage, including searching for and removal of restricted meats, plants, seeds, and so forth, is made. These articles when found are seized and generally destroyed. Because of reduced funds in recent years, baggage inspections have been seriously reduced. The Animal Research Service budget for fiscal year 1957 requested $856,000 to be used by Customs to restore previously established standards of baggage inspection at maritime ports and airports. At the same time, $163,000 additional funds have been requested to strengthen the technical quarantine service at ports of entry. Such meats are potentially dangerous because most of these sausages are made from ground beef, pork, and veal -- seasoned, dried, and not cooked. The danger lies in the fact that a lot of our people would disdain eating such a product, and when the friends and neighbors have gone they throw it in the garbage can, and there is the grave danger of the herds of raw garbage feeders becoming infected.

With the introduction of the airplane into the handling of livestock movements, we are faced with graver problems than we ever have been in the past. Most shipments coming from Europe, Asia, and Africa would take from 15 to 40 days by boat and, in some instances, would have knowledge of disease prior to the arrival of the ship and could properly control the arrival of the animals. However, since July 1, 1955, we

have received at the port of New York about 88 percent of the poultry importations by air and about 67 percent of the horse importations by air, and numbers of cattle and zoological animals -- the flight time of which is about 12 to 14 hours. This, you will understand, adds to our responsibility as exposed animals might not have had an opportunity to develop symptoms of a disease prior to the landing, but still would be infected unless we had some adequate quarantine facilities.

We are encountering considerable difficulty with the various research establishments that are using serum as a base for numbers of their experiments which they receive from foreign countries and, of course, these must be held up until we are satisfied that the serum used was not of ruminant or swine origin, or has been heated to a temperature which was safe for introduction into this country.

You might be interested in knowing that there have been times at the port of New York when over a million wet salted cattle hides have been imported during a month's period from infected countries and these must be handled off the ship, across the pier to sealed cars, trucks, and lighters, and sent to tanning establishments that are operating under the supervision of Animal Inspection and Quarantine for disinfection of such hides and skins.

Quarantine and regulatory personnel are kept informed of the presence and the distribution of animal and plant diseases in the various foreign countries. This information determines to some extent the decisions that are made for issuance of permits for importations of animals and plants.

Health certificates or inspection certificates are required for importation of animals, plants, seeds, bulbs, and so forth, brought into the country. Upon arrival of such shipments from foreign countries by boat or air, inspection is made to determine the presence or absence of diseases or pests. This service is conducted in cooperation with the Customs Service, the technical work being done by representatives of the plant and animal quarantine branches of the Agricultural Research Service. Many classes of nursery stock are released only after fumigation. Animals and poultry may be placed in quarantine for a period of time sufficient to be assured that they are free of communicable animal diseases, or exposure thereto.

DISEASES CAN GAIN ENTRANCE AND SPREAD

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U. S. Department of Agriculture
Washington, D. C.

An absolute protection against the entrance of foreign animal diseases into this country is a virtual impossibility; however, all available precautions must be taken to prevent the introduction, accidentally or by design, of such diseases. We are all familiar with the fact that the Federal Bureau of Animal Industry was organized to eradicate a so-called foreign disease - contagious bovine pleuropneumonia - and with the fact that Federal import restrictions have been of incalculable value in helping to keep foreign diseases out of the United States. There will always exist the threat of the accidental introduction of foreign animal diseases. It must be remembered that in addition to this threat there is also the possibility of deliberate introduction. One is inclined to wonder why, during the last generation, our enemy hasn't made use of this important weapon which is certainly at his disposal.

As veterinarians, we know that diseases often do the unexpected. The outbreak of foot-and-mouth disease a few years ago in Canada is a good example of this and is also a classical example of what might occur in this country. In this case, the disease appeared in a most unexpected location - in a very remote locality in the very heart of Canada, in an area where, perhaps unconsciously, we just don't look quite so hard for the unusual. Bluetongue is a good example of a disease that didn't "follow the book" and consequently went undiagnosed in this country for many years.

One of the principal problems in dealing with foreign animal diseases has been the lack of readily available information pertaining to them, together with the lack of experience in handling and in diagnosing them. Thus, we must become better acquainted with foreign or exotic diseases and be in a better position to recognize them if they should gain entrance into any part of the United States. The prompt recognition and the prompt reporting of the disease, of course, is a primary requirement for successful control and eradication. The sooner the disease is recognized and reported, the quicker action can be taken against it. The speed with which this action is taken will determine, to a great extent, whether the disease can be initially eradicated or whether a slower and more costly procedure of controlling it with the use of biologics and other measures will have to be used.

Recent experiences with vesicular exanthema illustrate how a highly communicable disease can remain somewhat localized and suddenly spread

throughout the country. The map on Page 28 shows that this disease spread into 42 States. This was due, to a great extent, to our livestock-marketing practices and the rapid means of transporting livestock and products to and from marketing centers. These factors make the control of such diseases extremely difficult in spite of our increased technological knowledge. Disease travels as man and his animals travel - as they travel faster and farther, the diseases affecting them do likewise. An illustration of this is shown in the diagram on Page 29 showing the possible manner in which scrapie spread throughout the United States.

Perhaps we should make reference to a word we hear more and more frequently in veterinary circles: EPIZOOTIOLOGY. Almost any epizootiologist has his own definition of this term but, for practical purposes, we might simply say that epizootiology is the study of the nature, etiology, and spread of animal diseases. By means of this study, we arrive at what appears to be a practical means of control and eradication of a particular disease. The fact that scrapie has an incubation period of 18 months to 3 years, or longer, makes it a particularly difficult disease to keep out of this country and to eradicate when it has gained entry.

In order to effectively control and eradicate animal diseases in the face of our marketing practices and the rapid movement of animals by rail and by truck, it soon becomes obvious that we must all band together and pool our knowledge and efforts if we are to enjoy any degree of success.

The necessity for united action in our disease eradication efforts is illustrated, I think, in the diagram on Page 30 showing how scrapie was discovered in this country. Since 1947, scrapie has been diagnosed in 42 flocks in this country. The top bar on the graph shows that of the 42 infected flocks, the disease was discovered in 18 (or about 43 percent of them) through tracing movements of sheep to or from suspicious or infected flocks or through subsequent routine inspection of such flocks. This illustrates the definite need for an organized program and plan in disease eradication.

The second bar shows that in the case of 10 (or about 25 percent) of the infected flocks, the disease was found and reported by practicing veterinarians. The local practitioner, with his experience and knowledge of the usual diseases in his community will, in all probability, be one of the first to realize that he may be dealing with an unusual condition.

The third bar indicates that another 25 percent of the infected flocks were discovered by the owners' seeking aid from regulatory officials. To me, this clearly illustrates the necessity of having a good public information program so that the industry will know to whom to turn for

assistance with their disease problems. Liaison and a working relationship with the livestock industry must be maintained. This same thought is apparent in the lower bar, which indicates that in the case of 4 flocks (or about 10 percent of them) scrapie was discovered when the owner consulted veterinary institutions in an effort to find a solution to his problem.

Here again we reap the benefits of a cooperative effort, this time showing the important part played by veterinary colleges in the diagnosis of disease and in disease eradication and regulatory programs. The veterinary college, of course, plays a multiple role in that not only do they aid the owner directly, but they furnish diagnostic counsel to the veterinarian and assist in his training by sponsoring veterinary short courses, etc. They also have an opportunity to instruct the student veterinarian in the principles of regulatory work throughout his academic training.

Our emergency disease eradication organization, if it is going to be of much practical value, must incorporate the full cooperation of the livestock industry, the practicing veterinarian, educational institutions, and regulatory authorities and must be carried to all related livestock groups. One of the best ways to do this is by showing films and other visual aids in conjunction with orientation meetings and other gatherings. We have some excellent films on both domestic and foreign diseases. To be fully effective, these films must be shown on a broad local level.

An example of how a foreign disease can enter this country and remain undetected, or at any rate undiagnosed, is illustrated on Page 31 which shows bluetongue as reported in the United States from 1948 to 1955. Here we have a disease that was not detected earlier, possibly because the mortality in this country has so far been a great deal lower than that reported in South Africa. In this particular case, we have a disease with a relatively low mortality that is widespread in certain areas of this country. However, we must be alert for the appearance of more virulent strains of the virus and be ready to take appropriate action should they appear.

We have a somewhat similar situation in regard to Newcastle disease. We are fortunate in that we seldom have the highly fatal form of Newcastle disease, in spite of the fact that the condition is not uncommon in our poultry. When the highly fatal form of the disease is discovered here, we must make every effort to stamp it out. In this connection, about January 20, 1956, a disease appeared in a laying flock in El Paso County, Texas. The owner indicated the birds seemed to have a "cold" and aureomycin was added to their feed, but they continued to get worse and started dying about January 23. By

January 28, about half the flock had died and the disease had spread to two neighboring flocks. The birds in the first flock were still showing only respiratory symptoms with rapid death losses. By February 6, 106 birds out of the flock of 114 had died, and 7 of the 8 remaining showed some paralysis or nervous symptoms. In the next few days another owner lost 20 out of 25 birds, one lost 90 out of 100, and one lost 10 out of 11 birds. In the latter flock several pheasants also died. Two other owners also experienced heavy losses.

One owner had about 40 birds located only 8 yards from the flock first affected. This flock had been vaccinated against Newcastle disease in November 1955 and in January 1956. As of February 14 this flock had not suffered any losses, except that two birds had shown slight nervous symptoms.

Here again we see the benefits of cooperative disease eradication. Specimens were sent to Dr. J. P. Delaplane of the Poultry Diagnostic Laboratory at Texas A & M College, who confirmed the clinical diagnosis of Newcastle disease. In the meantime, Federal and State veterinarians were sent to the El Paso area to determine the extent of the condition and to put control measures into effect. Fortunately the outbreak was not widespread and the majority of the larger poultrymen had practiced vaccination. Also fortunately, El Paso is not a large poultry-producing area and poultry and poultry products in this country tend to move toward the infected area rather than away from it.

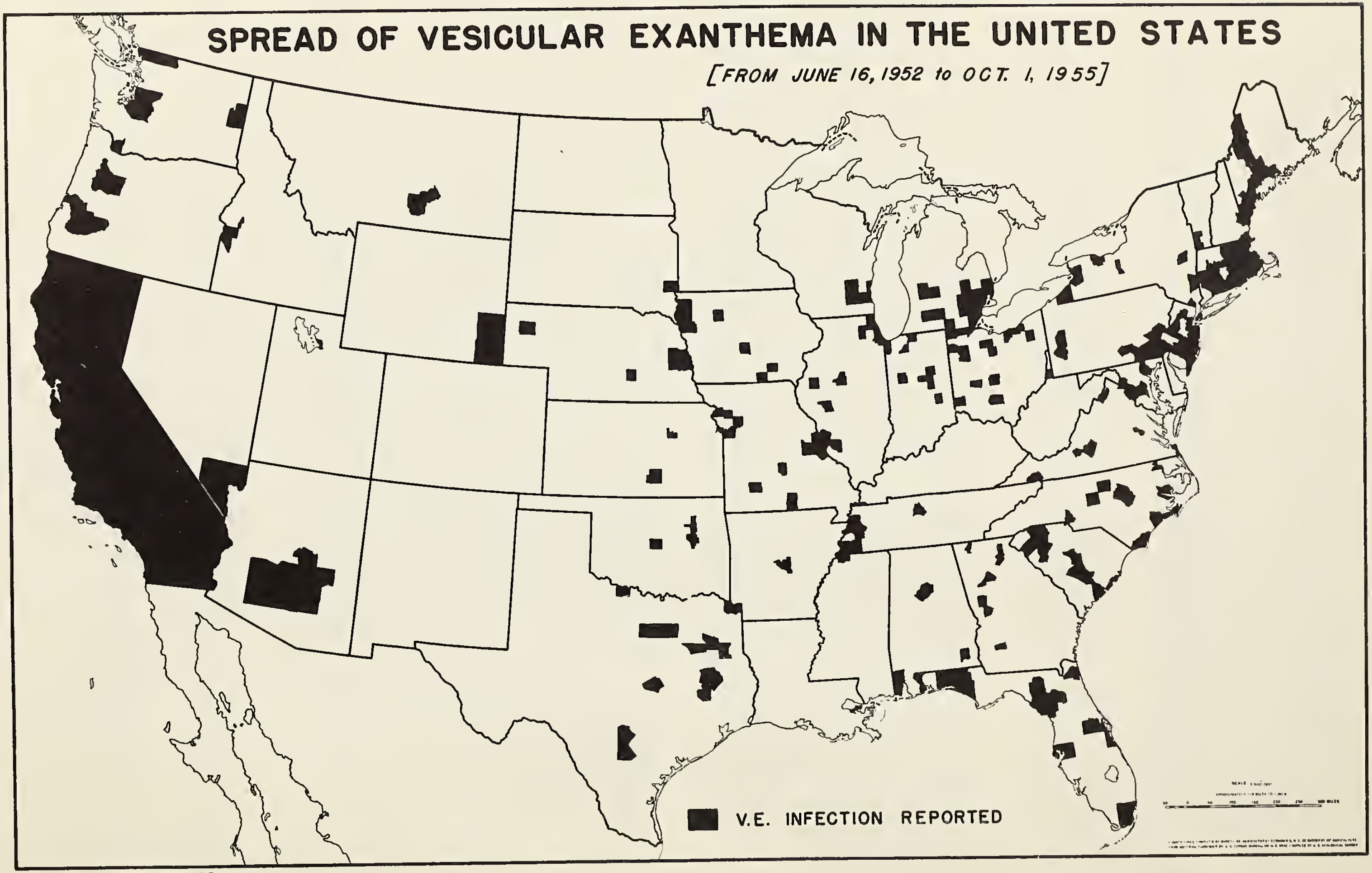
The control measures consisted of a vaccination program, along with good sanitation, for flock owners with healthy birds. On infected premises the dead and infected birds were burned or buried and covered with lime. The pens and chicken houses were cleaned and sprayed with a cresylic disinfectant. Feed dealers were instructed not to put chicken feed in used sacks. The poultry plants in the area were contacted and operators advised about the outbreak of Newcastle disease. They promised to cooperate and report any cases brought to their attention. Only two of the poultry plants handled live poultry. These plants were cleaned and disinfected. Owners of infected flocks were advised not to restock until April and to be sure to vaccinate against Newcastle disease. All veterinarians in the El Paso area and the County Agent were contacted, either in person or by telephone, and advised of the disease and the control measures that were being taken to prevent its spread.

This is the third wave of virulent Newcastle disease reported in the El Paso area since 1948. The disease has also been reported in Mexico.

In conclusion, foreign animal diseases or more destructive types of the diseases which we now have, can gain entrance and spread. Our best defenses against them are precautions to prevent their introduction

and well organized disease eradication programs and emergency disease eradication programs to handle them should they appear. We must pool our efforts and incorporate the full cooperation of all available agencies and persons in order to successfully defend ourselves against such diseases.

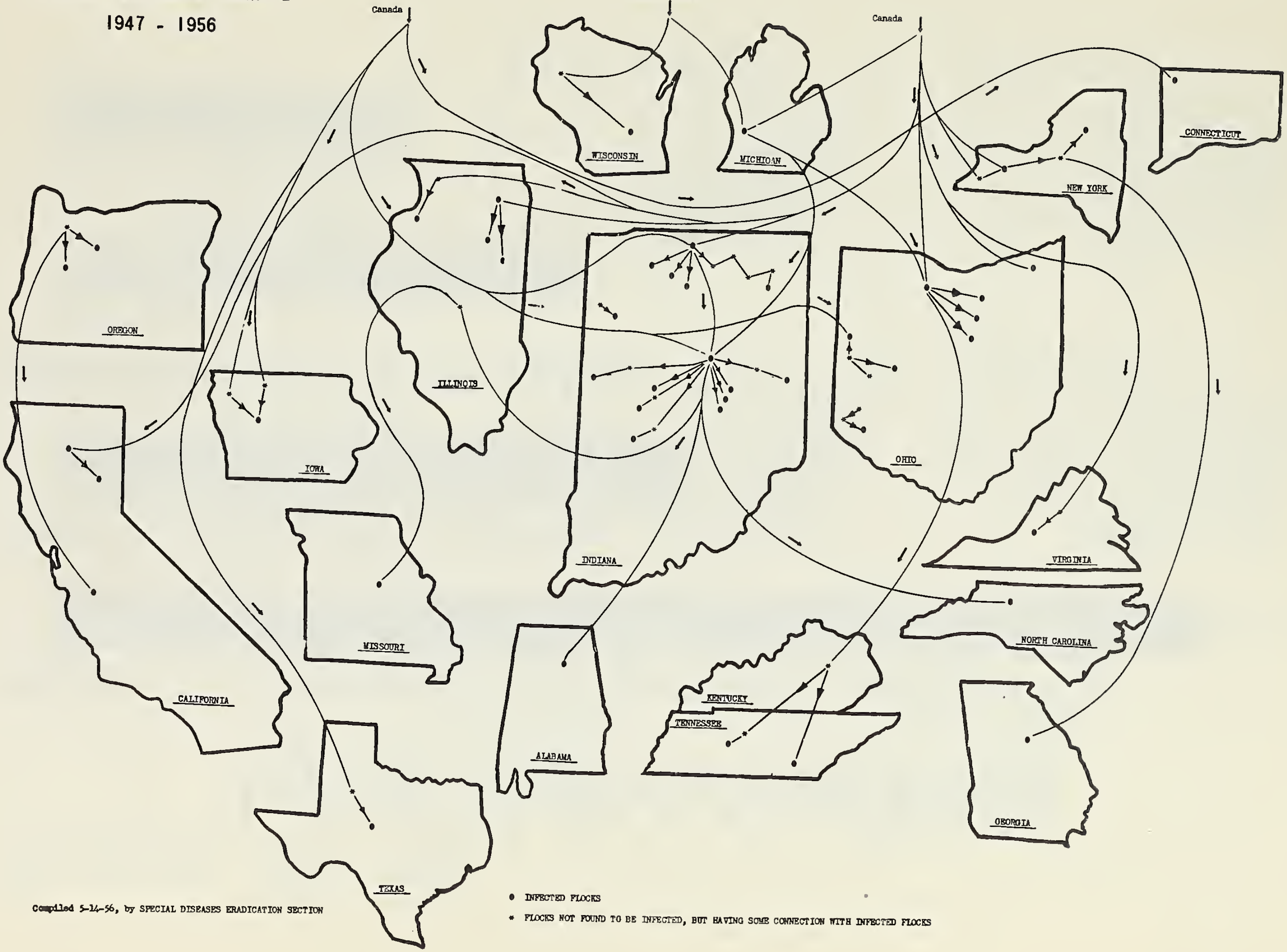
I believe there is much to be gained from personal contacts. We must carry - in the physical sense - our program to each other and down to the owner himself.



U.S. DEPARTMENT OF AGRICULTURE

POSSIBLE SPREAD OF SCRAPIE

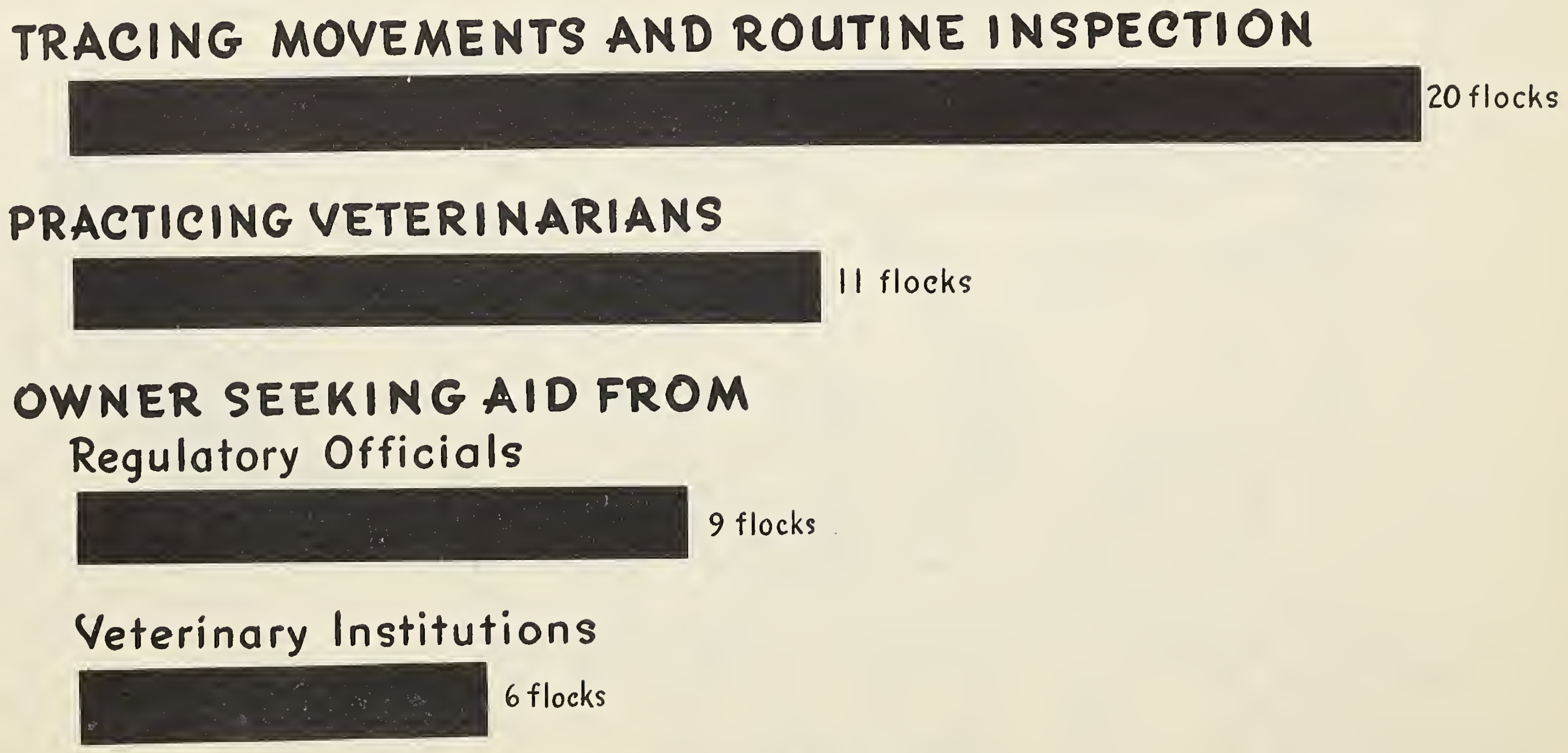
1947 - 1956



Compiled 5-14-56, by SPECIAL DISEASES ERADICATION SECTION

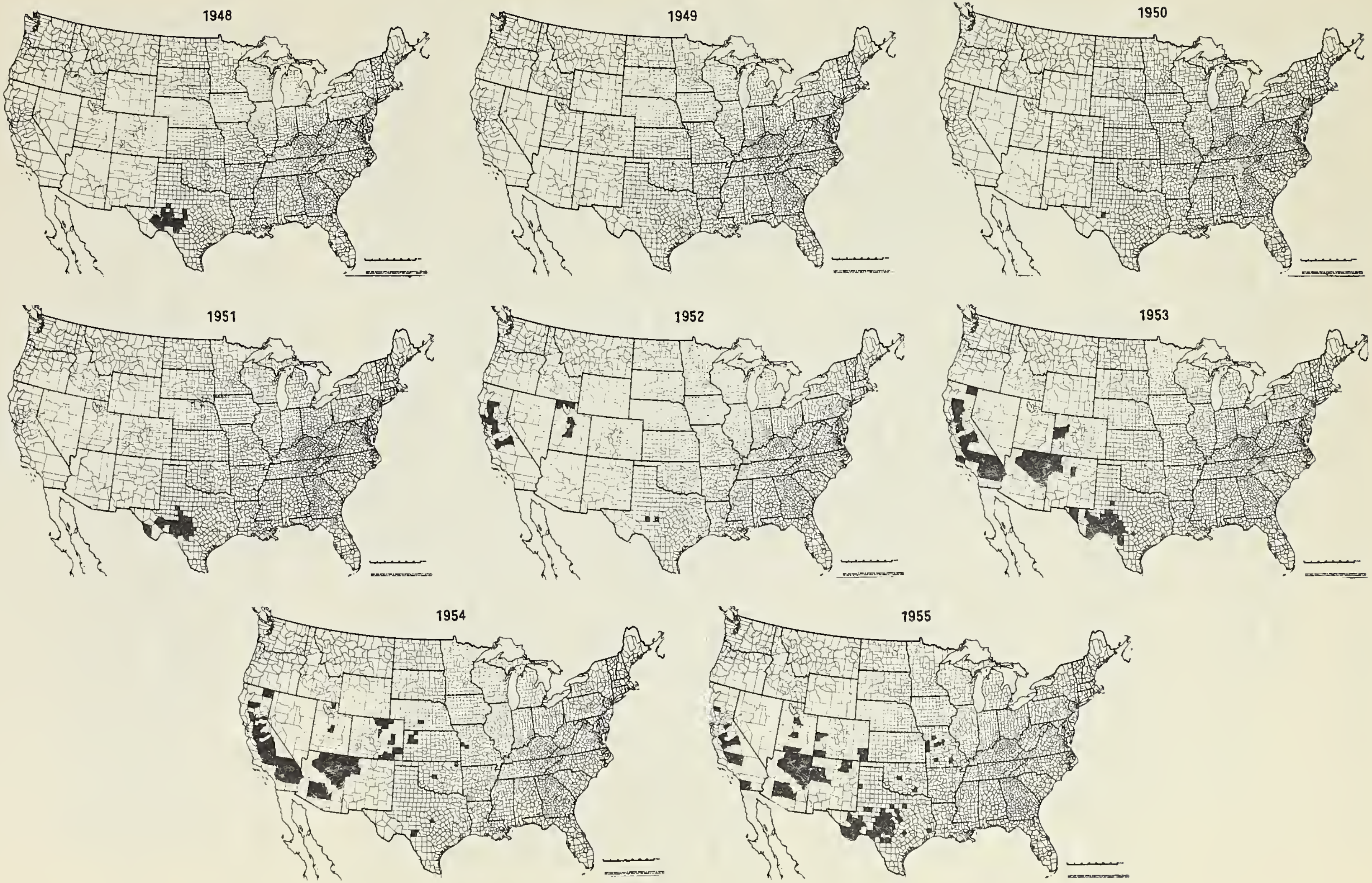
Scrapie, 1947-56

HOW DISCOVERED



DATA AS OF MAY 9, 1956

BLUETONGUE REPORTED IN UNITED STATES



THE NEED TO REPORT FOREIGN DISEASES EARLY

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We are all familiar with the topic "The Need for Reporting Animal Diseases Early". The addition of the word "foreign" in the title adds even more importance to this need.

From reading articles, posters, etc., on diseases such as cancer, tuberculosis, and so forth, in the human field, we see that they are having a similar problem. In veterinary medicine, however, it appears to be even worse, as we quite often realize that veterinarians are not called to treat the animals until the owner has used every means available to him without favorable response. It is encouraging to note, though, that due to veterinarian-client relationship this problem is being reduced.

In addition, to continue to encourage livestock owners to report the disease early, this paper is emphasizing the need of having veterinarians report them to their regulatory officials <u>early</u>. We all realize the value and necessity for immediately reporting any outbreak of animal disease. It is important from a diagnostic standpoint, and certainly it is a major factor in controlling the spread of diseases, thereby minimizing the loss from such outbreaks.

Plans and programs for dealing with foreign animal diseases should include, above everything else, every effort to prevent them from becoming established, and finally to eradicate them. This is a principle that should be kept in mind at all times.

The first thought that one dwells on when he reviews this principle is "How can these diseases become established?". The reasons can be grouped into (a) delayed diagnosis, and (b) livestock marketing.

<u>Delayed Diagnosis</u>

(1) The owner fails to report the condition until it is out of hand.

(2) Very few of these foreign diseases when first observed show typical textbook symptoms which makes their recognition difficult.

(3) Because of similarity of symptoms and lesions found in diseases already present in this country, the diagnostician will tend to ignore the possibility of the condition being a foreign disease.

(4) In some cases the diagnostician is not aware that the symptoms are those observed in foreign diseases.

(5) Indifference - "It cannot happen here" attitude.

(6) Veterinarian hesitates to report condition due to possible adverse effects on client relationship.

A classic example of delayed diagnosis in our own country could be the 1914 outbreak of foot-and-mouth disease. Animals suspicious of having foot-and-mouth disease were reported in August of 1914, but a positive diagnosis was not made until October 15, 1914. As a result, on October 19, 1914, two counties in Michigan, and two counties in Indiana were quarantined, but by the last of November quarantines were needed in twenty States and the District of Columbia.

A comparison can be made with the outbreak of foot-and-mouth disease in Canada. Investigations showed that the owner was treating some of his sick animals in the early part of December 1951. The owner reported the condition a week or two later when his animals did not respond to treatment, but an official diagnosis was not made until February 1952. In the interim, the disease was spreading to other herds.

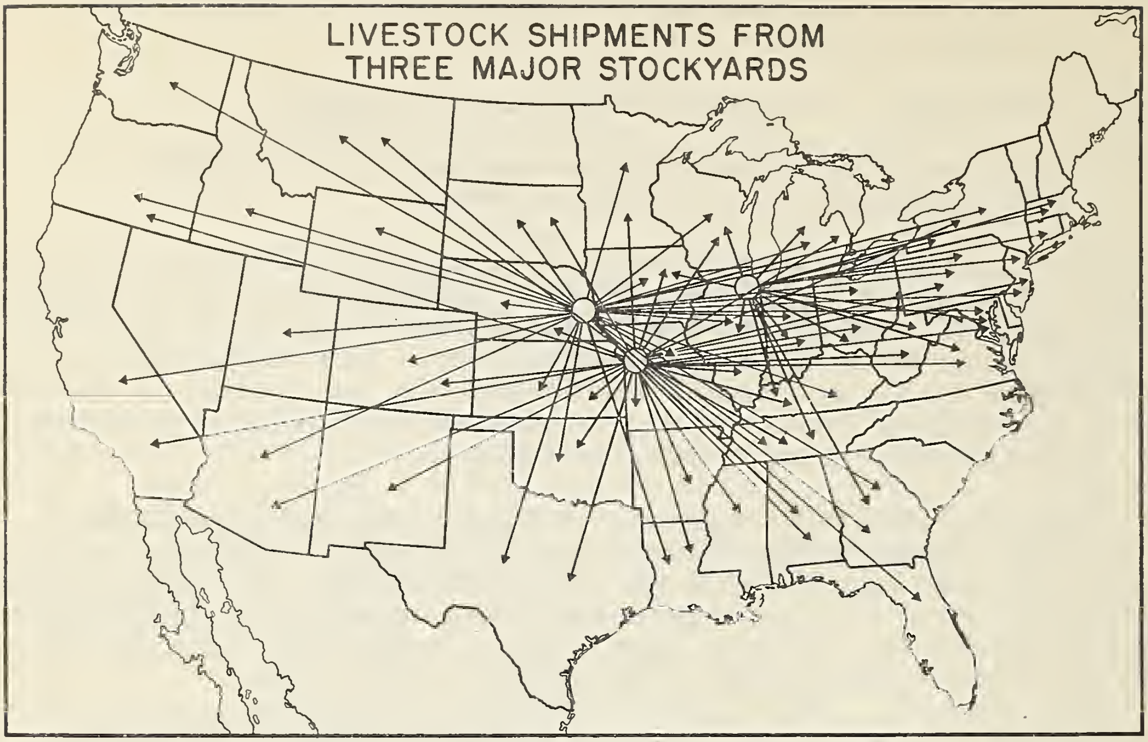
In Mexico, a condition which was believed to be vesicular stomatitis was reported in a newspaper article November 18, 1946. An investigation was carried out and a diagnosis of foot-and-mouth disease was made December 15, 1946, and later confirmed December 26, 1946. During this interim very little was being done to curb the spread of the disease, and by January 1947 the disease was reported in nine States. By May 1947, it was reported in 16 States and the Federal District - an area that involved about 17,000,000 animals.

In all three cases authorities report that the lesions were atypical, that they resembled those seen in necrotic and mycotic stomatitis, and that the disease was not spreading like foot-and-mouth disease should. Isn't there good reason to believe that this could happen again when very often we see reports of necrotic and mycotic stomatitis being clinically diagnosed?

Livestock Marketing

Our Intricate Means of Marketing Livestock

(1) For example, consider the shipments from three major stockyards and the States to which these markets normally ship livestock (see illustration on Page 34). These markets receive livestock from almost as many states as they ship to and are only three of



48 within our country that have Federal inspection. Therefore, in order to visualize the complete picture of this transportation pattern, one would have to multiply these shipments by many times to include movements from other yards in the country, such as stockyards not having Federal inspection, auction and sales markets, etc.

(2) The intensive production of our animals, the concentrated and extensive movements of these animals from their point of birth to sales markets, to feed yards, back to sales markets, and then to slaughter, provide an opportunity for most communicable diseases to become widespread in a very short period of time.

The account of the spread of the 1914 outbreak of foot-and-mouth disease was that at about the same time that quarantines were placed on counties in Michigan and Indiana, suspicion existed of the infection being in the Union Stockyards at Chicago. Later the diagnosis was confirmed. The disease was further disseminated from large eastern stockyards.

If foot-and-mouth disease could spread to 22 States and the District of Columbia in 1914, how many States would it spread to today if it got the same start?

In Mexico the routes that animals and people used were the avenues by which the disease spread. The amazing feature relative to the spread was that most movements of animals within the infected area were by foot. Can you imagine how the rapid means of livestock transportation in this country would lend itself to an accelerated spread of an exotic disease?

Discussion

We have not had a major foreign disease in this country for years, and most of our livestock industry does not realize how it would affect them if one did appear. If everyone in the industry understood the effects that one of these diseases could have, then it would not be difficult to obtain the cooperation of all.

Every veterinarian, when he sees a disease that resembles any of these exotic diseases, should feel that it is his patriotic and professional duty to report it immediately to the regulatory official. It is evident that this is not always being done, or we would not be getting reports of clinical diagnoses of vesicular stomatitis, necrotic stomatitis, or mycotic stomatitis. In such cases, the veterinarian would not be satisfied until he had called in a specially trained diagnostician and eliminated the possibility of its being foot-and-mouth disease.

Not to report a suspicious case of foot-and-mouth disease in England is punishable by a jail sentence. Because of the importance of the livestock industry to England in its economic structure, they fully appreciate the value of reporting diseases early.

Delay in identifying diseases, in determining the extent of infection, and allowing them to spread to many States also allows opposition to eradication to develop. This results in a great cry "Let's Live with It" from those who do not realize the seriousness of the situation.

It should not be the policy to wait until the veterinarian has finally reached a diagnosis before he reports any such conditions to his regulatory official. It should be reported to him as soon as the veterinarian is suspicious that it may be a foreign disease.

Getting owners of livestock to report their sick animals early is one field that needs a great deal of continued attention. This is a vital point in any animal disease control program. This is a point on which each livestock owner in this country has an obligation to his fellow livestock raisers. It is not a stigma on him when his herd is placed under quarantine, but rather that he is doing a very noble deed in preventing other premises from becoming infected from his livestock.

Every farm organization in the country should stress the value of owners reporting suspicious animals promptly to their State regulatory officer.

Better relationship between the regulatory official and all veterinarians in the State is absolutely essential to encourage this need of early reporting. Not until every veterinarian and every member of the livestock industry promptly brings to the attention of their regulatory officials any suspicious foreign diseases will we feel satisfied that adequate measures are being taken to report these diseases early enough so that we may prevent them from becoming established.

By now it should be quite plain that everything should be done to keep foreign diseases from becoming established. The first step in this direction is to report any suspicious case early.

AVAILABLE FACILITIES IN THE UNITED STATES FOR EMERGENCY DIAGNOSIS OF ANIMAL DISEASES

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Federal and State livestock sanitary officials who are charged with the prevention, control, and eradication of diseases of animals have long been concerned with the adequacy of diagnostic facilities in the various States to serve the needs of the livestock industry. Their concern is not only with laboratory facilities to aid in the diagnosis of diseases of domestic importance, of which there are many, but also for foreign diseases should they gain entrance into this country.

In reviewing the foreign animal diseases under discussion one is impressed with the similarity of their symptoms and lesions to several diseases existing in the United States. For example, the resemblance of rinderpest to Indiana virus diarrhea or mucosal disease is striking and has caused no little concern. Fortunately, it can be reported that both serum neutralization and cross protection tests conducted in Canada show that none of the conditions in the mucosal disease complex now existing in this country are in any way related to rinderpest. Likewise, African swine fever resembles American hog cholera in many respects but the diseases are immunologically dissimilar. These and other examples of similarity in foreign and domestic diseases could result in masking of a serious exotic disease under the guise of a native infection, thus permitting widespread dissemination of a dangerous disease if not recognized as such. An early and definitive diagnosis is of utmost importance in such circumstances. This would not only permit quarantine where indicated but would also prevent undue alarm if the condition were found to be an atypical form or increase in virulence of a domestic disease. The necessity of prompt differential diagnosis of suspected foot-and-mouth disease from other vesicular diseases well illustrates this point.

While Federal quarantine regulations on imported livestock and poultry and other inspection procedures tend to minimize or even prevent the entrance of foreign diseases into the United States, absolute protection is almost impossible. Whether the introduction be accidental or deliberate in the case of biological warfare, the free movement of livestock in this country could result in widespread dissemination of a disease in a comparatively short time. A control and eradication program then becomes more complex. The United States Department of

Agriculture has an enviable record in the eradication of foreign diseases that have gained entrance into this country in the past. Contagious pleuropneumonia, foot-and-mouth disease, and fowl plague have been eradicated every time they made temporary invasions. Constant vigilance must be exercised in keeping out these diseases as well as other very devastating and dangerous animal diseases.

The ways and means of preventing or combating any particular disease may depend to a large extent on our knowledge of its characteristics, manner of spread, and previous experiences with it. These or other factors may determine the means of control and eradication of a disease. Serious new animal diseases gaining entrance into the United States and Canada are eradicated by the slaughter method. The all-out effort put forth by the U. S. Department of Agriculture in preventing the entrance of foot-and-mouth disease from Mexico into the United States was a notable achievement. It is also an unprecedented example of a successful cooperative effort between two adjoining countries to control and eradicate a highly communicable disease which existed in one, and at the same time preventing its spread into the other.

Realizing the potential of such a threat to our animal population, the Federal Civil Defense Administration has delegated to the Department of Agriculture the responsibilities for defense against biological warfare against animals. In order to be better prepared to cope with occurrences of new or unusual disease outbreaks, the Department's Agricultural Research Service invited a group of outstanding poultry pathologists representing different sections of the country to come to Washington, D. C., to discuss the various aspects of the problem. While this group was primarily interested in how exotic poultry diseases could best be prevented, detected, and eradicated, the same general principles could be applicable for diseases of large animals. The conference resolved into several committees to make specific recommendations for future planning. It was pointed out that while the diagnosis of most of the large animal diseases can be made in the field, diagnosis of poultry diseases for the most part required laboratory assistance.

In order to evaluate the diagnostic laboratory facilities available in the United States, one committee recommended that a personal contact survey be made in each State on a regional basis. The country was divided into six principal regions and an area coordinator for each was designated to work with the Agricultural Research Service to carry out the committee's suggestion. In this survey of laboratories, special inquiry was made with regard to their fields of special study, personnel, equipment, and other facilities for disease diagnosis. A summary of the regional coordinators' survey revealed that there are now in the United States 93 laboratories, other than Federal and commercial, operating under the supervision of State Departments of

Agriculture or the Schools of Veterinary Medicine or Departments of Veterinary Science in the State Colleges or Universities, as the case may be, in the different States. All the laboratories reported doing some poultry diagnostic work, but less than 20 percent, are sufficiently staffed and equipped for special diagnosis of foreign poultry diseases or domestic poultry diseases of an unusual nature. This suggested the need for a central laboratory in each of the different areas to facilitate closer cooperation between laboratories and to function as referral centers. A key diagnostic laboratory and an alternate was tentatively designated by the regional coordinators in each region based on their available facilities for particular diagnostic procedures not common to all laboratories. The collaborators indicated that many of the same diagnostic centers selected for poultry could also serve in a similar capacity for the diagnosis of diseases of livestock.

The following laboratories with alternates, suggested by a committee on poultry diseases, would seem to qualify as regional centers in each area:

<u>Northeastern States Area</u>

Dr. Erwin Jungherr, Coordinator
Department of Animal Diseases
University of Connecticut
Storrs, Connecticut

> Department of Animal Diseases
> University of Connecticut
> Storrs, Connecticut
>
> Department of Veterinary Science (Alternate)
> University of Massachusetts
> Amherst, Massachusetts

This area is comprised of Connecticut, New York, Maryland, Rhode Island, New Hampshire, Maine, Pennsylvania, Vermont, Delaware, New Jersey, and Massachusetts.

<u>Southeastern States Area</u>

Dr. E. P. Johnson, Coordinator
Animal Pathology Section
Virginia Agricultural Experiment Station
Blacksburg, Virginia

Poultry Disease Research Laboratory
Virginia Polytechnic Institute
Blacksburg, Virginia

Department of Animal Pathology (Alternate)
University of Kentucky
Lexington, Kentucky

This area is comprised of Alabama, Tennessee, Kentucky, South Carolina, Georgia, West Virginia, Florida, Virginia, and North Carolina.

North Central States Area

Dr. Carl A. Brandly, Coordinator
College of Veterinary Medicine
University of Illinois
Urbana, Illinois

Iowa Veterinary Diagnostic Laboratory
Iowa State College
Ames, Iowa

Animal Disease Diagnostic Laboratory (Alternate)
University Farm
St. Paul, Minnesota

This area is comprised of Illinois, Indiana, Iowa, Kansas, Michigan, Minnesota, Missouri, Nebraska, North Dakota, Ohio, South Dakota, and Wisconsin.

South Central States Area

Dr. John P. Delaplane, Coordinator
School of Veterinary Medicine
Texas A & M College
College Station, Texas

School of Veterinary Medicine
Texas A & M College
College Station, Texas

Livestock Sanitary Board Diagnostic
Laboratory (Alternate)
Baton Rouge, Louisiana

This area is comprised of Texas, Mississippi, Oklahoma, and Arkansas.

Northwestern States Area

Dr. E. M. Dickinson, Coordinator
Department of Veterinary Science
Oregon State College
Corvallis, Oregon

> Poultry Disease Laboratory
> Oregon State College
> Corvallis, Oregon
>
> Western Washington Experiment
> Station (Alternate)
> Puyallup, Washington

This area is comprised of Washington, Montana, Wyoming, Utah, Idaho, Oregon, and Colorado.

Southwestern States Area

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This area is comprised of California, Nevada, Arizona, and New Mexico.

The diagnostic and research activities of the Federal laboratories located in the East Wing of Agriculture's Administration Building were suspended because of hazards to the health of those engaged in the work. This action was taken by the Administrator of the Agricultural Research Service on recommendation of three widely-known research workers outside the Department, who made an inspection of the laboratories in which the work has been carried on for about 50 years. The investigation committee found the physical facilities in the laboratories lacked modern safeguards, nor could they be reconstructed to protect the laboratory workers and other personnel in the building against possible exposure to tuberculosis, anthrax, and other communicable diseases under study. Apparently, this action was taken more as a precautionary measure as the health record in the laboratories over a period of 50 years has been very good.

The order to discontinue operations has seriously curtailed the diagnostic work as the handling of diseased tissues and work with live

cultures of dangerous organisms was no longer permitted. Several research projects were similarly affected. Lack of safe space at the Animal Disease Station to continue the examination of fresh tissues has limited our examination for the time being to histologic diagnosis of specimens received in formalin fixative. These include specimens received from regulatory workers in the field and those submitted from the Meat Inspection Branch. Fresh specimens received for diagnosis will have to be directed elsewhere until other facilities are provided. Serologic tests for dourine and glanders on imported equine stock and on vesicular stomatitis and vesicular exanthema will be continued at the Animal Disease Station at Beltsville. The production of Brucella antigen was not affected by the suspension order and will continue to be produced at Beltsville. However, the order did affect the production of tuberculin which was made in the Department's Administration Building for over 50 years. Because no space was available to continue its manufacture, it was necessary to arrange for tuberculin production with a qualified biological concern. Since the Department's protocol for the production of tuberculin is required in the contract, a product equal to that prepared in the Federal laboratories is to be expected. The diagnosis and research on bluetongue in sheep will be continued at the Animal Disease Research Laboratory, Denver, Colorado.

Work on the mucosal disease complex in cattle is being carried out by the Division of Veterinary Science at Purdue University. Facilities for the differential diagnosis of these diseases are available upon request from regulatory officials, but to a limited extent. This project is in part supported by Federal funds. Rhinotracheitis in cattle is being studied at the Veterinary School, Colorado A & M College, Fort Collins, Colorado. Diagnostic facilities for this disease are also available there upon request of livestock sanitary officials. Federal funds have also been allotted to aid in this project.

As regards vesicular diseases, the U. S. Department of Agriculture provides in each State a specially trained diagnostician to assist in investigations of suspected or reported vesicular diseases. In such instances, animal inoculation tests to establish the diagnosis must first be made by the diagnosticians on the premises where outbreaks occur. If the condition proves to be either vesicular stomatitis or vesicular exanthema on initial field trials, specimens from other cases occurring in the same general area can then be shipped by air to a Federal laboratory for typing of the virus. The preparation and shipment of such specimens is the direct reponsibility of the field diagnosticians. Serum from convalescent or recovered cases can later be submitted to the laboratory for the complement-fixation or serum-neutralization test to determine previous exposure to one vesicular disease or another in case of question.

As a result of the laboratory suspension order in the East Wing, the Administrator of the Agricultural Research Service has developed plans to provide adequate new physical facilities with required safety features, for the conduct of all phases of the Department's animal disease research and diagnostic work including space for an expanded biologics testing program.

Knowledge of any disease is one of the essential tools with which to control it. The more information that can be gained on exotic diseases, the better position we will be in to cope with emergency situations should they arise in the United States. As a means of gaining first-hand experience to aid in a better understanding of foreign diseases, the United States Department of Agriculture and the Department of Defense have from time to time sent laboratory workers to other countries to study and work with the more serious exotic diseases where they exist. These men bring back to us valuable information and illustrative material on diseases that can be discussed at regional meetings. These special foreign assignments have provided a number of trained diagnosticians who are available as consulting specialists. These persons can be called on in emergencies, upon request of State and Federal officials, to aid in establishing a definitive diagnosis without delay.

Another manner of obtaining diagnostic aid for exotic diseases is to send suspected material to laboratories in foreign countries where the disease is known to exist. As an example, identification of bluetongue virus in sheep in the United States was made in South Africa by Dr. R. A. Alexander. He was later invited by the U. S. Department of Agriculture to come to this country to discuss with State and Federal livestock sanitary officials and laboratory workers methods of diagnosis, control, and other aspects of bluetongue.

Mention has already been made of the assistance given by the Canadian government in conducting serum neutralization and cross protection tests to differentiate the group of mucosal disease complex in this country from rinderpest. In making the cross protection tests possible, the Department's Agricultural Research Service made the necessary arrangements and provided funds to truck two recovered cases of mucosal disease, Indiana viral diarrhea and rhinotracheitis to Canada for these tests.

In turn, by agreement, our laboratory at Plum Island will make their facilities and personnel available to the Canadian Department of Agriculture to conduct tests on any suspected cases of foot-and-mouth disease.

One of the principal means of diagnosing these diseases is the ready availability of diagnostic agents for the diseases about which we are

concerned. These should consist of (1) representative strains of causative agents that could conceivably be introduced, (2) diagnostic sera, (3) specific antigens essential for the diagnosis of various diseases, and (4) a tightly controlled central repository for safe keeping of exotic infective agents. In properly equipped laboratories, the tools for handling emergency diagnosis are essentially the same as for the routine diagnosis of an existing disease. In tightly controlled laboratories, the necessary viruses for emergency diagnosis could be made available to them from a central repository under proper safeguard. Another diagnostic aid, and often an effective one, is a distinctive microscopic picture seen in some of the exotic diseases. The histologic picture coupled with clinical and autopsy findings may contribute to a proper diagnosis or perhaps serve to rule out the disease under suspicion. Certainly, histologic examination is an effective tool in differential diagnosis in our domestic diseases. Sets of histologic preparations of a number of exotic diseases are available at the Armed Forces Institute of Pathology for comparative study.

Differential Diagnosis

1. Rinderpest

   Presumptive diagnosis can be made based on clinical features, gross and microscopic pathology.

   A definitive requirement: cross protection tests or serum neutralization test. These serological procedures can be accomplished in Canada.

2. Mucosal Disease

   The clinical, gross and microscopic changes are differentially helpful but a positive diagnosis requires cross protection tests. To date, neutralization tests have not been accomplished. Duration of immunity is lasting.

3. Indiana Viral Diarrhea

   Clinical, gross and microscopic changes are presumptive but again a confirmed diagnosis requires cross protection tests. Indiana viral diarrhea produces a convalescent immunity lasting only about 4 months.

4. New York Viral Diarrhea

   Very difficult to distinguish from Indiana viral diarrhea. On the basis of clinical or gross pathology, it remains to be seen

whether microscopic changes are actually differential. So far the anti-serum obtained from cases of New York viral diarrhea have not proved satisfactory for neutralization tests. So cross protection tests are required to differentiate between this group of mucosal type diseases. Preliminary work in tissue culture with Indiana viral diarrhea suggests that it may provide a useful tool for diagnosis.

All of these cross protection tests are accomplished by challenging known convalescent immune cattle with the homologous and heterologous virus which might be involved.

Controls of such cattle are necessary.

5. Bovine Pleuropneumonia

This is a frequently benign type of bovine pneumonia which is difficult to distinguish from other types on clinical grounds. The herd history may be helpful. The complement-fixation tests are the best means of diagnosis in the live animals. Gross pathology is characteristic and diagnostic; the production of broad interlobular septa and walled off, encapsulated sequestra are characteristic and never bilaterally symmetrical. Histologically it provides a distinctive lesion.

6. Rhinotracheitis

Rhinotracheitis stands alone on the basis of gross and microscopic pathology, in comparing it with other diseases in this group. It might, however, be readily confused on clinical grounds with necrophorus infection and shipping fever. It is the only one of these diseases which primarily involves the trachea. Cross protection tests may be accomplished and the virus has been established on tissue culture which can be neutralized with immune serum.

7. Bovine Malignant Catarrhal Fever

Bovine malignant catarrhal fever is primarily a disease of the turbinates with characteristic involvement of the eyes and frequently of the CNS. Such changes as occur in the digestive tract and the respiratory tract may be thought of as extensive from the inflammatory process in the head.

Unfortunately there are no specific serologic means for diagnosis and very little work has been done with the agent.

It has been transmitted to rabbits. The diagnosis is primarily dependent upon clinical evidence, gross and microscopic pathology.

8. Teschen's Disease

Clinical suspicion should be aroused by febrile disease of swine producing symptoms arising from the CNS which include irritability, tremors, convulsions, flacid posterior paralysis. There are no gross lesions of significance. Microscopically the most constant changes are degenerative ones in the ventral horn of the spinal cord. Here the changes are very similar to those seen in polio. Changes also occur in the brain, particularly degenerative changes in the cerebellum. These may also be lymphocytic perivascular and meningeal infiltrations. The virus may be transmitted to susceptible animals from infected brain and spinal cord. In such transmissions studies animals immune to hog cholera should be included to distinguish from the neurotropic forms of hog cholera.

9. African Swine Fever

The principal problem in African swine fever is distinguishing it from hog cholera. In general, the lesions of African swine fever are much more severe than in cholera. Most outstanding are the hemorrhagic lymph nodes which may look more like hematomas than lymph nodes. Interlobula edema of the lungs in African swine fever, congestion and edema of the gall bladder and a generally more severe destruction of vascular endothelium are noted in African swine fever. Cross protection tests in which animals immune to hog cholera are susceptible to African swine fever should always be done to confirm the diagnosis.

10. Rift Valley Fever

Young mice are susceptible and the infection in mice may be prevented by known immune serum so quantitative serum neutralization tests may be accomplished in mice to confirm a presumptive diagnosis made on clinical and gross pathologic grounds. For laboratories set up for the work, complement-fixation tests are much preferred for diagnosis to eliminate the hazard to laboratory workers in having to conduct necropsies and serum-virus neutralization tests.

RESUME OF THE MEETINGS OF THE REGIONAL COORDINATORS AND LABORATORY REPRESENTATIVES HELD IN CONJUNCTION WITH THE REGIONAL CONFERENCES ON FOREIGN ANIMAL DISEASES

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At each of the six regional meetings held under the auspices of the Agricultural Research Service, United States Department of Agriculture, in March and April 1956, representatives from practically all diagnostic laboratories in the various States operated by State Departments of Agriculture, Agricultural Experiment Stations, and veterinary colleges were in attendance. In addition, State and Federal regulatory officials from each of the States were present at the meetings. Also, many State and Federal public health veterinarians attended.

The meetings were presided over by the poultry disease collaborators from the various regions who were previously designated by the Agricultural Research Service to counsel and advise the Department on the prevention of foreign poultry diseases and their control should they make their appearance in the United States.

The original purpose for designating these collaborators on foreign poultry diseases was as outlined above. The first meeting of the collaborators was held in November 1953, and several valuable suggestions were made at that time which were acted upon, such as extending the length of quarantine for certain birds imported into the United States. The collaborators stressed the importance of early recognition, confirmation of diagnosis and proper handling of suspected foreign diseases as well as domestic diseases of unusual occurrence. Another recommendation was that a complete survey be made of the diagnostic facilities. This was done through questionnaires and by personal visitation of the collaborators in their respective regions.

At a later meeting held June 20 and 21, 1955, in Washington, these reports and surveys were reviewed and certain laboratories in each region were designated as primary and alternate reference laboratories for assistance in the diagnosis of poultry diseases, particularly those suspected of being exotic.

Duties of the Laboratories:

1. Each designated laboratory with outstanding personnel and facilities will serve as the primary focus of diagnostic effort in the case of unusual diseases.

2. Because of probable initial lack of capacity to cover the entire field and all possible problems, the central laboratory will serve also as a clearing house for integrating special examinations and studies and in diverting material to several other laboratories within the region especially qualified for and interested in certain specific diseases.

3. Inter-regional effort, as well as that between the region and Federal and international agencies, may be effectively integrated through the central laboratory of the region.

When facilities being utilized by Agricultural Research Service, in Washington, D. C., and Beltsville, for diagnostic purposes had to be abandoned because they were considered no longer adequate for the conduct of this type of work, each collaborator was asked whether or not the reference system of diagnosis might be extended to all animal diseases. The collaborators replied that the survey of the various diagnostic facilities revealed that with few exceptions, the same laboratories designated as regional or reference laboratories for poultry diseases were also involved in the diagnosis of other animal diseases and could serve in the same capacity for both. This met with the approval of the laboratory representatives at each of the six regional meetings when presented for discussion.

It was pointed out at each meeting that it was not the intention of any of the State diagnostic laboratories to burden the regional or alternate reference laboratories with problems which they were able to cope with themselves. On the other hand, suspected or foreign disease problems requiring special diagnostic facilities should be referred to them when help is desired. It was emphasized that specimens should be referred with the approval or knowledge of both the State veterinarian and Federal official in charge in the State of origin and the recipient State, and that the reference laboratory should be contacted before specimens are submitted.

The nature of the suspected agent should govern the manner of packing and shipment in interstate movement. Every precaution should be taken to prevent leakage of the container. In some instances, it may even be advisable to deliver the specimens by personal messenger depending on the nature of the infectious agent. These procedures are suggested to serve as a recommended chain of referring suspected disease tissues.

The collaborators pointed out that it was also a function to keep the various regions informed of any unusual disease outbreaks and also on occasion help would be sought from laboratories in the various regions specializing in certain diseases or groups of diseases. An example is the aid of the veterinary school at Colorado A & M College, Fort Collins, Colorado, in cases of suspected bovine rhinotracheitis, or the Division

of Veterinary Science, Purdue University, in the case of viral diarrhea or mucosal disease, after preliminary screening has been made in local laboratories. Likewise, other disease conditions can be channeled in a similar manner to regional or other reference laboratories when emergencies arise.

The collaborators commented on the activities of the regional reference laboratories to serve as repositories for museum and library headquarters. They cited the Newcastle and fowl pest kodachrome collection furnished by the Agricultural Research Service as an illustration of a beginning in this respect. Kodachrome collections of other disease conditions will be furnished the reference laboratories as they become available.

The Agricultural Research Service has indicated that when reference laboratories are called on by State or Federal regulatory officials to assist in the diagnosis of exotic or unusual domestic disease conditions, that funds will be made available for the purchase of additional animals and hiring extra help in carrying out the project; also, that Federal personnel when needed will be assigned to assist in the work.

Recommendations by Coordinators, and Laboratory Workers:

1. Furnish each State maps of both reference and alternate regional laboratories.

2. Establish working relationships with the United States Public Health Service for cooperation where zoonoses are involved.

3. Furnish all laboratories with definite information for contacting coordinating personnel in case of emergencies (telephone numbers, name, etc.).

4. As kodachrome slides of representative animal diseases and titles become available, provisions for loan or even individual copies of such materials should be made available to the States; further that such materials include reprints and descriptive information not readily available.

5. Officials in charge of all laboratories furnish the Animal Disease Eradication Branch, Agricultural Research Service, Washington, D. C., copies of their monthly diagnostic reports.

6. A comprehensive report of this information with special items of interest be furnished by the Agricultural Research Service to all cooperating laboratories.

7. Each diagnostic and research laboratory be classified as to diseases with which they are equipped to work.

8. A complete telephone directory of responsible officials in charge of laboratories, State livestock sanitary officials, veterinarians in charge of field stations, and Washington officials be published and kept current by supplements.

Conclusion

Since the meetings, personnel in the Animal Disease Eradication Branch have been working toward activating the recommendations given above. Material will soon be coming to laboratories and State and Federal regulatory officials in accord with the above recommendations. As rapidly as possible, the other recommendations will be carried out.

EMERGENCY ANIMAL DISEASE CONTROL AND ERADICATION PROGRAMS

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In the disease programs which are carried out on a cooperative State-Federal basis, certain factors determine the type of program to be used for the control and eradication of specific diseases. For example: We have the diseases that are indigenous to the United States and those of an exotic nature which are introduced. Among the indigenous diseases we can refer to tuberculosis, brucellosis, hog cholera, scabies, etc. Examples of exotic diseases are foot-and-mouth disease and rinderpest.

The decisive factor, which determines whether or not a program of control and eradication is necessary from a State or Federal standpoint, is its economic effect on the livestock industry. There are some diseases of a highly contagious nature which affect the economy so fast that the livestock industry demands that something be done about the disease immediately. This demand for action is almost simultaneously felt by the State and Federal regulatory officials. An example of this is vesicular exanthema which has been reported in 42 States since June of 1952. In the other extreme, we have diseases such as leptospirosis or anaplasmosis, which could very well be having the same effect on the affected species of livestock that the effect of vesicular exanthema had on swine. However, the nature of the spread of these diseases is not as spectacular.

The support of the industry for control and eradication of diseases such as leptospirosis or anaplasmosis is much slower. It develops from herd owner to herd owner, then to an area, then to the State, and does not reach national attention until the effects accumulate to produce the necessary interest. Examples of programs that develop to such proportions just described are tuberculosis and brucellosis.

Animal disease programs are not the dreams and the sole wishes of regulatory officials. Rather, the regulatory officials are advisers to an industry that today has a value of over 12 billion dollars. The regulatory officials do not dictate to this industry as to which diseases are going to be eradicated, but recommend to them those which they feel are of such economic importance that it is to their advantage either to have them controlled or eradicated.

Many times we have heard criticism from individuals within our own profession against the old fashioned method of test and slaughter, or slaughter and burial as a means of eradicating a disease. Some say, "You spend millions of dollars testing and slaughtering animals when biologics could be used", and that "such practices of slaughter and burial are not based on technical science". However, some of these slaughter programs have resulted in the complete eradication of the disease at a far less cost than any other means available. From a technical standpoint, the disease has been completely eradicated within a shorter time than biologics would have been able to do it.

Do not interpret this as a stand against the use of biologics, because in some diseases we recommend them, such as in bluetongue, calfhood vaccination for brucellosis, and equine encephalomyelitis, etc.

When biologics reach a stage in which they can be used as effectively in eradicating diseases as slaughter methods have, then we will be only too glad to replace the slaughter methods with them.

Naturally, before a program of control and eradication is approved, it must have a sound basis. The basis for a sound program is (1) the support of the livestock industry; (2) knowledge of disease (causative agent, how it is perpetuated and spread); (3) accurate method of diagnosis; (4) adequate measures available to control the disease; (5) adequate authority to carry out the program; and (6) adequate funds.

In foreign diseases, the potential dangers that may be encountered, should they become established in this country, are considered. Also, the means of handling them, should they be reported, are given a great deal of study, such as the discussions that are being held at this meeting.

In a disease such as foot-and-mouth disease, on which we have had a great deal of experience in eradication, there is no question as to the proper method for eradication. Perhaps in other diseases, such as rinderpest, African swine fever, etc., the same method used in foot-and-mouth disease would be applied. In other foreign diseases, not considered to be as damaging as these, circumstances may largely determine the program. For example, any foreign disease reported in this country, in which the extent is limited, should be completely eradicated. However, when similar diseases do become established, consideration has to be given to whether rapid, complete eradication is economically feasible; also, whether the information on the means of controlling and eradicating the disease is available to develop a suitable program.

From experience gained in Mexico and other countries, we have developed a plan which, if properly organized, will work because it has worked under more adverse conditions than we will ever find in this country - we hope.

You recall when foot-and-mouth disease first struck Mexico, it was five years before it was considered that the outbreak was eradicated. In contrast, there has been one other outbreak since then which was brought under control and eradicated and confined to a localized area in one State. This job of eradication was done within a few months. What was the difference? Early reporting and an organized operation ready to move at a moment's notice.

The first move in attempting to develop a plan of organization was to discuss it with those who would have to use it, namely, the regulatory officials of the country. Last year a series of meetings was held throughout the country for the purpose of discussing this plan with the regulatory officials. A chart (see Page 56) showing the recommended organizational set-up to handle emergency disease operations was proposed.

The plan is one of action. The method of instituting the plan is to select individuals in advance who may serve in key capacities, should an animal disease emergency develop in any State. The key man would be an individual selected to head the emergency disease operation, under the supervision of the State Veterinarian and the Animal Disease Eradication Branch veterinarian in charge. The designated individual would be more apt to give the operation his undivided attention, since he would know that in case of an outbreak he would have the burden of responsibility. The person selected would have more time to spend on plans in the interim between now and when an outbreak may occur than would the State and Federal veterinarians in charge and this type of planning requires considerable attention.

Realizing that the two top officials, and even a veterinarian whom they might select to head such an operation, cannot come up with all of the answers, it is suggested that a staff be selected. The staff would consist of an information officer, administrative officer, supplies and maintenance officer, and an operations officer to form a type of staff. This group would meet periodically and develop plans. For example: What would they do if they had to employ 500 people tomorrow? Where would they get the money to pay indemnities? How would they handle the information to the public? How would they get the vehicles for transportation of the additional personnel being hired? Who would assign veterinarians for inspection and diagnostic purposes? Who would determine the areas to be placed under quarantine? Who would appraise infected and exposed animals? Who would work on the marketing problems? This can go on and on and on.

As you can see from the chart, the planning goes beyond the staff down to field units. The field unit would consist of a group who would actually handle the eradication of the disease within an area of the State. There may be many of these field units operating within a State.

Just as the various States have been developing a staff, persons in the different fields within the U. S. Department of Agriculture have been selected to serve on the Branch staff. They are making certain recommendations to the Chief of the Branch, and steps are being taken to overcome obstacles that may delay action when a foreign disease strikes. For example, steps are being taken to have special authority already available in case an emergency develops. Meetings have been held with various agencies within the Department of Agriculture requesting their cooperation in aiding these emergency disease programs in any way that they may be called upon to do by the Branch veterinarian in charge and the State Veterinarian. This means organizations such as Extension Service, Forest Service, Agricultural Marketing Service, Commodity Stabilization Service, Soil Conservation Service, etc., throughout the country are ready to give them their support.

Reviews have been made of the organizational charts which have been submitted by the various State and Federal officials, and these charts have been discussed with the regulatory officials. It is hoped that we can keep our planning along realistic lines so that they will be effectual when they are put to use. We must guard against plans that would not be practical even though they may sound good in theory.

The program for control and eradication would be based on the following lines:

1. Report any suspicious condition resembling an exotic disease promptly to your State or Federal regulatory official.

2. State or Federal regulatory official will place quarantine on premises until diagnosis is made.

3. Veterinary inspection of adjoining premises will be made to determine extent of infection.

4. All shipments of livestock to and from the premises will be checked for the source of the disease, as well as the possibilities of spread.

5. Test material will be sent to designated laboratories for diagnosis, if necessary.

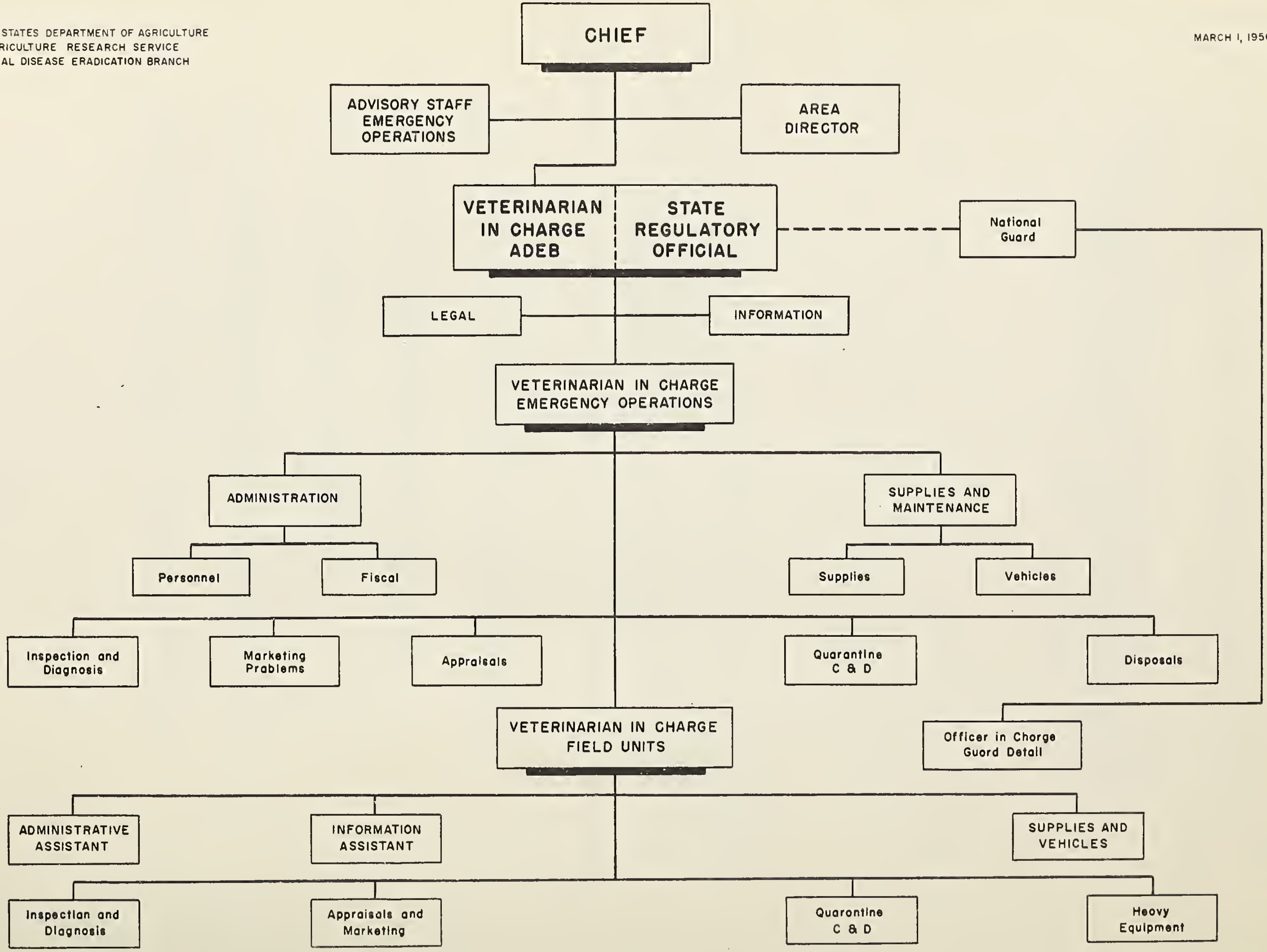
6. Once diagnosis is made, if it is an exotic disease, the veterinarian in charge will place into effect emergency operation.

7. Emergency operation will consist of quarantine; inspection and diagnosis; cleaning and disinfection; slaughter, if necessary; vaccination, if appropriate; and other measures, depending on the condition involved.

PROPOSED
STATE, FEDERAL EMERGENCY DISEASE ERADICATION ORGANIZATION

UNITED STATES DEPARTMENT OF AGRICULTURE
AGRICULTURE RESEARCH SERVICE
ANIMAL DISEASE ERADICATION BRANCH

MARCH 1, 1956



STATE-FEDERAL COOPERATIVE ANIMAL DISEASE ERADICATION

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The principle of State-Federal cooperation has been accepted in this country as essential to the eradication of communicable livestock diseases since 1884 when the Federal Bureau of Animal Industry was established by an Act of Congress. Prior to this time, the States had attempted, individually, to control and eradicate such diseases within their borders. This manner of operating might have continued longer had it not been for a situation that developed in 1843. During that year, a "ship cow" affected with contagious pleuropneumonia was purchased by a New York City dairyman. From this animal, a chain of infection started which spread throughout the New England area, along the Atlantic Seaboard and, eventually, as far west as Illinois. The failure of individual States to limit the spread of highly contagious and fatal diseases of this nature demonstrated the need for coordinating control and eradication efforts under a central agency, having authority extending across State lines.

In reviewing the early history of animal disease control efforts in this country, it is interesting to read about the difficulties encountered at that time and to note the similarity between earlier conditions and those we encounter today. The following quotations taken from the recorded history of events leading to the creation of a Bureau of Animal Industry, reflect earlier public thinking along these lines.

> "It was generally felt that under existing conditions the efforts to expel the invader (Contagious Pleuropneumonia) would not be successful and the livestock industry must continue indefinitely to endure the losses and to be harassed by the rigid quarantine restrictions imposed by various States for their protection or as retaliation.
>
> "The best-informed livestock owners and veterinary authorities came to realize that a national direction of the activities for the extermination of the malady would overcome the worst and most discouraging features which prevented the efforts of the individual States from being effective. The traffic in affected cattle would cease, the work would be more thorough and energetic, because the inspectors would not be directly or indirectly dependent upon the good will of the interested cattle owners for their positions, and the excuse so often presented of the inability of the States to pay for the diseased cattle would also be overcome."

In recognition of this situation, funds were appropriated by Congress for the fiscal year 1887 to purchase diseased animals. Maryland was the first State to cooperate under rules and regulations set up by the Bureau of Animal Industry for the eradication of this disease. Gradually other States began to cooperate and by 1892, after five years of concerted effort, the country was proclaimed free from contagious pleuropneumonia. A recorded statement made at the time is still appropriate: "If the States had been prepared with laws and funds, eradication of this disease could probably have been accomplished within one year ...".

The historical background of cooperative disease eradication in this country has been reviewed to show that cooperation between the States and the Federal Government has been considered essential for successfully combating contagious animal diseases since the first serious outbreak occurred, and has continued throughout the years as an accepted practice under our form of government.

In considering organized disease eradication in the United States, it must be remembered that all cooperative projects are State-sponsored. Uniformity in State programs has come about gradually through the adoption of standard practices recommended by the U. S. Livestock Sanitary Association. This organization of industry representatives, livestock regulatory officials from each State, research and educational workers, and Federal regulatory officials, meets annually to study the status of animal diseases in the Nation, review the programs for control of the common diseases, and make recommendations for improving control and eradication procedures. These recommendations constitute the basis for cooperative livestock disease control and eradication programs. The Animal Disease Eradication Branch of the Agricultural Research Service, has the responsibility for coordinating individual State programs along lines that will maintain cooperative animal disease control efforts at a level consistent with the needs of the national industry, facilitate movements of livestock interstate, and insure the confidence of foreign nations in the quality of our livestock and livestock products. All cooperative disease eradication work is performed under Memorandums of Understanding or "Agreements" executed by the appropriate State livestock regulatory official and the Chief of the Animal Disease Eradication Branch, which define the bases for cooperation and participation by the respective agencies.

With the development of a mutual understanding of what is necessary for effective cooperative efforts, these agreements have become more flexible in their provisions for State-Federal participation. The number of personnel, the amount and type of equipment, and the financial support provided by each may vary widely between States within limits considered necessary for each agency to perform those functions which are its particular responsibility. To a degree, this

is a departure from the earlier policy of fixed and equal particpation in all categories. Irrespective of the extent of State support, the Federal government is definitely concerned with disease problems wherever they exist. The maintenance of a healthy and successful livestock industry in each State contributes to the national economy and helps protect the industry of adjacent States.

Upon invitation of the State and completion of a Memorandum of Understanding covering each cooperative project, the Animal Disease Eradication Branch normally establishes an office in the capital city. The veterinarian assigned to the State as Veterinarian in Charge of Field Activities is responsible for working with his counterpart in the State service, usually the State Veterinarian, in developing effective programs designed after the recommended and generally accepted procedures for controlling and eradicating the diseases in question. The programs are planned in accordance with State-Federal financial support available for the work and in keeping with State laws and regulations governing these diseases. The Veterinarian in Charge is responsible for all Federal features of cooperative program planning and administration, and is assisted in this and the field supervision of program activities by an Assistant Veterinarian. The field organization is considered to be under the immediate supervision of the Assistant Veterinarian in Charge and/or his opposite number in the State organization. Close direction and work guidance is handled through "District" veterinary supervisors who are responsible for program activities being carried out in accordance with State and Federal policies.

The "District" supervisor, either a State or Federal veterinarian, is given responsibility for all cooperative activities within a designated portion of the State. This varies between States, depending upon the extent of program demands, but usually ranges between one-third and one-seventh of the area involved. These veterinarians are responsible to the Veterinarian in Charge and the State official, through their respective assistants, and supervise "District" operations through "Area" veterinarians. The latter are assigned a limited number of counties in which they work directly with the cattle owners and practicing accredited veterinarians in carrying out procedures incidental to actual testing and other field activities.

Under the present policy of implementing field operations, provision is made for accredited veterinarians to perform farm or ranch testing and vaccinating in a certain area on a unit fee schedule basis. In many instances, payment for these services is provided on a mutually approved basis. In other cases, the State may assume all fee testing expenses for a portion of the year with the Branch taking over payments for the remaining months, or the State and the Branch each will accept claims for the conduct of a particular service throughout the year.

In the case of tuberculosis, indemnities are shared, with the Federal government allowing a maximum of $25.00 for grade and $50.00 for purebred reactors, computed on one-third of the difference between carcass value and appraisal. In no instance can Federal indemnity exceed that paid by the State nor shall the total carcass value and State-Federal indemnity be more than the appraised value of the animal. Federal indemnity is available also in the case of brucellosis reactors identified in official cooperative work. However, it is not mandatory that the State participate in this feature of the program. Indemnity maximums are similar to those for tuberculosis and amounts are computed by the same formula of one-third the difference between carcass or salvage value and appraisal.

Herd and area quarantines required for the control of animal diseases are the responsibility of the State, but Branch veterinarians serving in an area capacity are commonly deputized by the State to lay quarantines in line with their over-all responsibilities.

Under the flexibility of operation permitted in State-Federal cooperative programs, there is considerable variation in the methods of record keeping. In all cases, however, it is essential for adequate records to be maintained in a manner that makes them readily accessible to both agencies and with as little duplication as possible.

So far, this discussion has dealt with the most common type of cooperative agreement, in which separate State and Federal administrations are maintained. Recently, two other forms of administration have been designed and put into operation on a limited basis in which there is a blending of administration and forces. The first of these provides for a single administrator of all cooperative programs and purely State and Federal responsibilities. Under one Director, State and Federal personnel operate as a unit, but otherwise are individually responsible to their respective agencies. The Director of all activities is a jointly paid employee who is equally responsible to the State Commissioner of Agriculture, or other governing body or official, and the Chief of the Animal Disease Eradication Branch. The only significant change in this type of organization is the single Director replacing the Federal veterinarian in charge and State regulatory official.

The other modification of the original system provides for the Federal veterinarian in charge to assume responsibility for all cooperative projects, such as brucellosis, tuberculosis, vesicular exanthema, scrapie, etc. Both State and Federal personnel operate under his supervision, with the State Veterinarian carrying the responsibility for other miscellaneous disease problems, quarantines, investigations, and commission sales control. The Federal Veterinarian in Charge remains a full-time employee of the Government under this plan, but is responsible jointly to the State and the Animal Disease Eradication Branch on all matters pertaining to cooperative work.

MEAT INSPECTION AND ANIMAL DISEASE CONTROL

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As a veterinary meat inspector, I welcome this opportunity to discuss our program as it relates to animal disease control. This subject takes on particular significance as we apply it more specifically to the responsibilities of the veterinary profession when veterinarians take on the duties of protecting the livestock population of this country against the ravages of foreign diseases. We in Meat Inspection are in position to have a full appreciation for those reasons why animal disease is one of the fundamental public interests.

Each year we inspect approximately 100 million food animals. We see a lot of sick animals and a volume of gross pathology that taxes the imagination. We know firsthand what animal disease can do to the livestock and meat economy of this country. Even with a reasonably healthy livestock population the losses from condemnation in federally inspected meat packing plants are enormous.

What is more important for the purpose of this discussion is our awareness of the part played by the system of livestock and meat distribution in this country in the dissemination of animal disease. Veterinary meat inspectors are so located in these systems as to promptly detect occurrences of disease in food animals as they are marketed for meat production. Recognizing this, veterinary meat inspectors have always been identified with those who are in a position to report occurrences of disease before it has an opportunity to become widespread. In order to function effectively in his position to detect and report occurrences of a foreign disease which might be introduced into this country, the veterinary meat inspector must be kept fully informed concerning such diseases, their characteristics, and the probabilities of their occurrence in this country. The system of reporting must be set up effectively and kept in continuous operation so that the veterinary meat inspector will know to whom a report is to be made and how to make it. Obviously, the detection and reporting of the occurrence of a foreign disease, while an important step, is only the beginning. To be of any significance, it must set in operation a control routine.

It is important to emphasize that meat inspection laws are not animal disease control laws. The meat inspection law is phrased so as to protect the consumer. As is the case with most all laws that affect

private interest, the legislators are careful to limit the powers of those who administer the law to a clearly defined objective. Under a meat inspection law, the veterinary meat inspector is not told that he might do anything which he considers to be a good idea for all concerned. On the contrary, he is told that he only has authority to condemn product which is unwholesome and unfit for human food. The standards which he applies in attaining this objective are clearly defined and internationally recognized.

For the purpose at hand, it is sufficient to say that these standards do not call for the production of a sterile product. Meat can be perfectly normal, healthful, wholesome, and fit for human food without being sterile. This is of particular significance when we consider viruses and organisms highly contagious for animals but of no hygienic significance for man. You know, of course, I do not mean that meat from sick animals is passed for human food. I do mean, though, that meat derived from animals in apparent good state of health and which meat appears normal macroscopically is passed for food and, while it is safe, wholesome, and meets all consumer demands, may be infectious for livestock.

If meat which is passed by a veterinary meat inspector as being normal and fit for food under authority contained in a meat inspection law is to be surrounded by controls to avoid its carrying contagion to livestock, power to accomplish this must be found in animal disease control legislation. How that power is to be brought to bear on meat or on animals ready for slaughter in a meat packing plant is a problem that can only be worked out and resolved by authority drawn from animal disease control legislation.

We in Meat Inspection are forced to be quite conscious of the importance of acting within the limits of legislative power. Every action we take affects substantial property rights of people. The courts quite properly are quick to protect anyone against the illegal action of an inspector. Obviously, a meat inspector who presumes to exercise powers beyond those contained in meat inspection legislation which he is administering is acting illegally unless additional powers are delegated to him. In fact, he may be held by the court to be personally liable in damages for his illegal act.

All of this is mentioned by way of emphasizing the importance of understanding the position occupied by the veterinary meat inspectors in this animal disease control picture. His position is an important one because he is located where he can do a lot of good. First, in the important role of early detection and reporting and second, where his responsibilities can be enlarged or extended by clear delegations under animal disease control legislation, his services can be invaluable.

Obviously, this latter cannot be left to chance or last minute expediencies. The veterinary meat inspector has been trained to operate within the law and we must expect that he will insist that any action that might be expected of him is fully supported by adequate legislative power passed on to him through clear and positive delegation. The animal disease control official can be sure that the veterinary meat inspector has a keen appreciation for his problems and wants to be part of the animal disease control team. He will, however, expect the animal disease control official to recognize the necessity for adequate legislative coverage for any action which might be expected to be taken by the veterinary meat inspector.

Many of you have had firsthand experience in how the veterinary meat inspector has been effectively integrated into the animal disease control team. The Vesicular Exanthema Eradication Program is a good example in this connection. In administering that program, the animal disease control official was faced with a viral disease similar to foot-and-mouth disease which had to be stopped in its tracks. Veterinarians everywhere had to be alerted to detect instances of the disease so that immediate diagnoses could be made to eliminate the probability of foot-and-mouth disease and at the same time institute such quarantines as would be effective to stop the spread of the disease.

The animal disease control official alerted the veterinary meat inspector and worked out with him a routine that would be effective for detection, reporting, and stopping the spread of the disease. The arrangement was for the veterinary meat inspector to call the animal disease control official by telephone if a suspected vesicular condition was detected at the inspected meat packing plant. When such a condition was detected by the veterinary meat inspector, he called the animal disease control official and described to him the findings in detail. The animal disease control official then decided what action was to be pursued with respect to the affected and exposed animals or the carcasses and byproducts of such animals.

The action usually taken by the animal disease control official was to quarantine the animals and immediately dispatch a diagnostician to the meat packing plant. The veterinary meat inspector by prior delegation of authority from the animal disease control official enforced the quarantine at the meat packing plant which required that the animals, carcasses, or byproducts be held until the vesicular condition was diagnosed. Upon receipt of the diagnosis, the animal disease control official decided on the appropriate disposition to be made of the affected and exposed animals, carcasses, and byproducts. If he decided

that the animals and product were to be required to be processed at the inspected meat packing plant to control the spread of the disease, the veterinary meat inspector continued the animals in quarantine and supervised their slaughter and processing in the plant. This would be adequate to assure that no product derived from these animals left the meat packing plant unless it had been subjected to temperatures adequate for destruction of the virus.

In summary, I want to point out that those actions of the veterinary meat inspector connected with holding the affected and exposed animals pending diagnosis and supervising the processing of the animals and their products to assure adequate heat treatment for the destruction of the virus were taken under authority delegated to him by the animal disease control official since such authority is not contained in meat inspection legislation. Plans for integrating the veterinary meat inspector into an effective specific animal disease control routine are easily made. The relative responsibilities are easily identified and the animal disease control official will find the veterinary meat inspector responsive to clearly defined delegation of special authorities. Of course, the animal disease control official must have the legislative authority in the first place if he is to delegate it and the arrangements should be clearly understood by both parties.

I hope that you will not be confronted with other emergencies like the outbreak of vesicular exanthema. But it is inevitable that similar situations will arise and I want to assure you that the veterinary meat inspector welcomes an opportunity to participate in an effective well-defined plan.

CONTAGIOUS BOVINE PLEUROPNEUMONIA

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Definition: Contagious bovine pleuropneumonia is a specific, infectious pneumonia of cattle caused by a pleuropneumonia-type organism called Asterococcus mycoides. It has a very long incubation period, produces a high mortality, is clinically difficult to diagnose, and in several countries, like Africa, it is of secondary importance only to rinderpest.

History: The disease has been known as a specific entity for over two hundred years. In the 1800's it spread from Europe to many other countries including the United States. By 1887 it had become so widespread and destructive in the United States that the Congress established the Bureau of Animal Industry in order to combat the disease. Through an intensive campaign of slaughter and quarantine the disease was eradicated from the United States by 1892, and it has remained free since. In about 1854, the disease was introduced into South Africa through the importation of a Friesland bull from Holland. The disease spread rapidly causing death in over 100,000 cattle within a 2-year period. From time to time it has spread over the greater part of Europe. The disease is presently distributed over much of the world, except for Western Europe and the Western Hemisphere.

Host Range: Pleuropneumonia is primarily a disease of cattle. Buffalo, goats, and sheep are apparently susceptible to artificial infection. The following species have been found non-susceptible: mice, rabbits, guinea pigs, horses, camels and swine.

Etiology: Asterococcus mycoides

Characteristics of the organism:

1. Growth in cell-free media with the development of extremely pleomorphic structures, including rings, globules, filiments and bazaar forms. Also, minute, filterable, elementary bodies or granules 125 to 250 millimicrons in size which are the minimal reproductive units.

2. The development in suitable solid media of characteristic growth which may vary from minute colonies of 10 to 20 microns to colonies as large as 600 microns in diameter.

3. The parasitic members of the group of pleuropneumonia organisms are capable of growth only in media containing a high concentration of serum protein and at temperatures above 30°C. The saprophytic organisms are capable of growth in media without serum protein and at temperatures below 30°C.

4. Some of the pleomorphism described for the organism has been due to distortion incurred in the process of staining. To avoid this the organisms have been grown in agar under a cover slip and stained in situ. All of the variations of the morphology during the growth cycle have not been firmly established. It is sufficient for our purpose to know there are numerous large filimentous and globular as well as the small spherical elementary bodies which are filterable.

5. The organism has been grown in a variety of media providing there was at least 10 percent serum protein in the media. Giemsa is a good stain for revealing the organism. The organisms have also been cultivated on the chorio-allantoic membrane of embryonated eggs, as well as in tissue culture.

Transmission: Under natural conditions the disease is contracted by inhalation of the aerosol produced by the coughing of infected animals. During the acute phase of the illness the virus will be present in the blood and infected tissues. Following encapsulation of the infection in the lung, virus may be present only in the walled-off tissues. If subsequently the tissue capsule should rupture with dissemination of the virulent contents throughout the air passages, the animal will again become a spreader of the infection. Consequently, latent carriers may harbor the infection for two or three years before they transmit the disease. Ingestion of the organism does not produce infection. Because of its very long incubation period, the disease may be transmitted widely before it is recognized. Likewise, carriers may carry the disease for months or years either latently or actively, causing wide dissemination of the virus while appearing quite normal. Because the disease is characterized by a pneumonia, it is difficult to distinguish it clinically from other types of pneumonia. The owner is likely to ignore a chronic cough, assuming it to be a minor infection rather than to become suspicious of bovine pleuropneumonia, especially when the animal remains in good flesh as these infected animals often do.

Transmission - Experimental: Daubney[1] transmitted the disease by the intrajugular inoculation of cultures mixed with 10 percent agar. The agar plugs became lodged as emboli in the lungs with subsequent

proliferation of the organism. More recently, transmissions have been accomplished by the exposure of cattle to high concentration of infected aerosols.

Incubation Period: The incubation period of this disease is from 10 to 120 days, but most clinical cases occur between 3 and 7 weeks.

Clinical Symptoms: A typical acute case of pleuropneumonia shows pneumonic symptoms which are clinically indistinguishable from those observed in non-specific pneumonia. It is also significant that a small proportion of the animals affected with this disease do not show any clinical symptoms and do not have a temperature rise although they may be infected and be capable of transmitting the disease. The temperature resulting from different individual cases is so variable as to be unhelpful in characterizing the disease. In the acute case, a cough occurs which is first dry and painful, later moist, and is followed by labored respiration with a grunting expiration. When the lung involvement is severe the animal may stand with the elbows out and headed into the wind with nostrils dilated showing respiratory distress. In the later stages there may be mucopurulent discharges from the nose and sometimes an edematous infiltration of the lower part of the chest. Weakness and emaciation may follow rapidly. Swelling of the joints is sometimes seen in young calves.

Pathologic changes: In typical cases the lung lesions of pleural pneumonia are unmistakable. In the ordinary acute case only one lung is involved. While the lung lesions may be bilateral, they are never symmetrical. This is an important point because some other diseases such as East Coast fever and hemorrhagic septicemia may show similar pneumonic changes but in those diseases the lesions are usually symmetrical and bilateral. The pleural cavity on the infected side usually contains a quantity of pleural fluid which may be blood-stained and contain strands of fiber. The infected lung is usually adherent to the thoracic wall. The involved lung tissue does not tend to collapse. It is usually firm and raised above the relatively normal adjacent tissue. Cut surfaces of the involved area show a marbled appearance; red or grayish areas of parenchymal tissue marked off by yellowish bands of thickened interlobular septa. These septa often show a beaded appearance due to the presence of distended lymph spaces. Areas of necrosis which occur during the acute phase tend to become encapsulated by a thick layer of fibrin which eventually becomes organized and numerous adhesions forms. The encapsulated portion which may be as large as an orange, is referred to as a sequestrum and although necrotic, may contain much of its original conformation. There is frequently a semi-fluid zone around the sequestrum immediately beneath the heavy connective tissue capsule. These walled-off areas may eventually dry up and leave merely scar tissue or they may become abscessed or rupture causing an acute flare-up of the infection. Most

of the animals die during the acute stage of the illness but a high percentage of those which survive until the lesion becomes encapsulated eventually recover. With some very virulent strains of the virus, mortality has reached 100 percent. Other strains have been found very mild, producing a low mortality. Histologically the lung appears as one would expect with broad bands of connective tissue dividing the lung parenchyma into lobules. The lobules contain alveoli that vary from normal to those that are completely consolidated. There is an infiltration of round cells around the blood vessels and bronchi and centered in small clusters within the interlobular septa. These round cells are predominately lymphocytes and plasma cells, with a sprinkling of monocytes and polymorphs. In the liver there is a round cell infiltration into the hepatic triads and some necrosis of individual liver cells adjacent to central veins. These necrotic liver cells are acidophilic and have dark pyknotic nuclei. In the spleen the germinal centers are enlarged. There is a decrease in mature lymphocytes; an increase in plasma cells, and an excess of blood cells and blood pigment.

Diagnosis: History, clinical symptoms, gross pathology, culture of the organism, and serological testing by the complement-fixation test are all means of establishing a diagnosis. The complement-fixation test will reveal late acute and chronic cases. In recovered animals there is a gradual decline in complement-fixing antibodies. Cattle inoculated in the tail develop a positive complement-fixation reaction within a few days but the test becomes negative within 6 weeks.

Immunization: A culture vaccine has been in use in countries like East Africa for many years. Prolonged artificial culture led to some attenuation of the organism. Production was accomplished by the subcutaneous inoculation of a calf with the attenuated culture. A few days later the calf was killed, skin removed from the involved area and the profusely edematous fascia was scraped and the infected lymph obtained. This infected lymph was used to inoculate cattle into the tip of the tail. It was too virulent for inoculation closer to the body and if done so, a severe local reaction occurred. Even when inoculated in the tail, the animals occasionally lost their tails. Immunization was not too successful with this vaccine and often resulted in the loss of highly susceptible cattle. Within the last four years a new method of immunization using an attenuated live virus vaccine prepared from an egg adapted strain has been developed in Kenya. With this vaccine a single subcutaneous inoculation has produced an effective immunity without undesirable reactions. It has several advantages over the old culture vaccine in that it is possible to store the vaccine dry for long periods, it can be produced in large quantities, and can more readily be tested for potency before use. Presently the most serious disadvantage is that the batches are somewhat variable in virulence.

Control: Control is dependent upon prompt diagnosis and the elimination of infected cattle. A plan which has been effective in freeing some large areas of the disease include the following provisions: alert veterinarians to the recognition and diagnosis of the disease. Report all positive and suspect cases to the central disease control authority. Destroy all positive cases. Conduct complement-fixation tests on all possible contacts. Destroy the positive reactors. Vaccinate the non-reactors. Quarantine the involved herds for 2 months and keep them under surveillance for an additional 6 months period. Two noteworthy characteristics of the disease which simplify the quarantine requirements are its host specificity and that transmissions occur only by direct contact.

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INFECTIOUS RHINOTRACHEITIS AND MALIGNANT CATARRH

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Infectious rhinotracheitis and malignant catarrhal fever are included because of some similarities to exotic diseases. Peculiarities of these diseases enable a differential diagnosis in most cases. Each disease will now be considered separately.

INFECTIOUS RHINOTRACHEITIS OF CATTLE

A contagious disease of cattle, infectious rhinotracheitis is characterized by inflammation of the upper respiratory system, and is caused by a virus. In Colorado, this disease was first recognized as an entity in 1950. Since that time, the incidence has increased annually until it is now the most important single disease of feedlot cattle.

Infectious rhinotracheitis has been diagnosed in Arizona, New Mexico, California, Utah, Wyoming, Colorado, Nebraska, Idaho, and Illinois. The possible existence in, or spread to, other states is recognized. Only cattle are affected both naturally and experimentally. Attempts to transmit the disease experimentally to sheep and laboratory animals have been unsuccessful. Under natural conditions, beef cattle being fattened, and dairy cattle are susceptible. To date the occurrence of the disease in range cattle not being fattened has not been reported. Experimentally, however, all types of cattle are susceptible. The seasonal incidence is higher during late fall and early winter months, although the disease occurs during all months in feedlots which are occupied through all seasons.

The cause, as indicated earlier, is a virus which is present usually in nasal secretions and exudates, and occasionally in blood of affected cattle. Experimentally, the virus can be transmitted to all types of cattle by intranasal or intratracheal inoculation as an aerosol. The experimental disease is mild and of short duration. Recovered animals resist reinfection temporarily at least. In feedlots the viral infection commonly is complicated by secondary bacterial infection of the respiratory system. Presumably the secondary bacterial infection causes more injury to the mucous membranes than does the virus. The bacterial infection may progress into broncho-pneumonia. *E. coli*, *S. necrophorus*, *Pasteurella*, *Staphylococci*, and *Streptococci* have been identified in lesions.

Symptoms develop rapidly and are characteristic. Body temperatures range from 105 degrees to 108 degrees Fahrenheit. Affected animals refuse feed and water and lose body weight rapidly. Fat animals may lose as much as two hundred pounds during the course of the disease. Frequently the muzzle is hyperemic and encrusted with exudate and desquamated epithelium. The nose shows mucopurulent discharge. Respiration is rapid and frequently progresses into noisy dyspnea and oral breathing. Coughing occurs throughout the course of the disease. Conjunctivitis is mild, but results in conspicuous lacrimation. Strings of saliva discharge from the mouth which contains no lesions. Diarrhea is occasionally present and pregnant heifers may abort. Leukopenia is not present, the course of the disease is of two types: first, the simultaneous affectation of most animals in the herd over a period of ten to fourteen days. The incidence is high and the mortality low. This type of course often develops in feedlots where the disease is occurring for the first time. The second type of course is sequential affectation of most animals in a lot over a period of forty to sixty days. Only a few animals are sick at a given time. Commonly morbidity is high and mortality low.

A survey of the feedlot cattle of Colorado has shown an incidence of approximately seventeen percent. Of those animals affected, approximately three percent died.

In summary, infectious rhinotracheitis of cattle is an acute inflammation of the upper respiratory system, without involvement of the mouth. Morbidity is high, while mortality is low. The disease is caused by a virus, often complicated by secondary bacterial infections.

MALIGNANT CATARRHAL FEVER

An acute infectious but non-contagious disease of cattle, malignant catarrhal fever is characterized by high fever, inflammation of the eyes and skin and by inflammation of the digestive, respiratory, and nervous systems and is caused by a virus.

Malignant cattarh occurs in cattle only. All breeds are susceptible. Animals from two to five years of age show higher incidence than other age groups, although only very young cattle appear to be not susceptible. Some studies of this disease have shown the incidence to be higher during spring and summer months. Cases occur, however, during all seasons. Geographically, this disease is widespread throughout the United States and occurs in other countries where cattle are produced in large numbers.

The cause of malignant cattarh as indicated earlier, is a virus. During early stages of the disease, the virus is in the blood and can be demonstrated in lymphoid tissues during later stages. Strong evidence has been advanced to show that sheep, although not susceptible to the disease, may act as a reservoir for the virus, and from sheep the virus may spread to cattle. In Africa, sheep and some species of wild mammals constitute a reservoir for the virus. The method of transmission has not been clearly established although the possibility of transmission by insect vectors has been suspected and not eliminated.

The symptoms of malignant catarrh may resemble several other diseases but usually present peculiarities sufficient to enable a differential diagnosis. Body temperature ranges from 106 to 110 degrees Fahrenheit. Early the blood shows leukopenia. Conjunctivitis and keratitis are severe and progress to opacity, ulceration, and sometimes perforation of the cornea. Inflammation of the ciliary body of the eye results in the accumulation of fibrin and blood in the anterior chamber. The blood and exudate are moveable and visible prior to opacity of the cornea. The muzzle becomes encrusted with desquamated cells and exudate. Early in the disease the mouth is hyperemic and later develops numerous small shallow erosions on the tongue, palates, and buccal mucosa. Copious mucopurulent exudate discharges from the nose. Breathing may become rapid and difficult. Diarrhea is common. Lymph nodes, especially of the cervical region, may be enlarged. Dermatitis with marked reddening of the skin may develop around the eyes, at the base of the horns, at the medial surfaces of the posterior limbs and near the external genitalia. Vulvitis and vaginitis may be encountered in female cattle and inflammation of the prepuce in male cattle. Excitement to the extent of fury with progression to depression may result from involvement of the central nervous system.

In summary, malignant catarrh is an acute viral disease characterized by inflammation of the eyes, skin, digestive, respiratory and nervous systems with low morbidity and high mortality. It occurs in cattle only.

INFECTIOUS INFERTILITY OF CATTLE

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Introduction: Sterility is a major problem in the cattle industry. This problem is receiving considerable attention by scientists in various fields and all the time new information is being added. And even though such important diseases as brucellosis are being mastered, the problem of infertility remains a major concern of cattle breeders and, in fact, one gains the impression that some diseases like vibriosis, trichomoniasis, and leptospirosis are causing more damage now than they ever did before. My purpose is not to discuss these, but another disease which seems to be gaining in significance. This is the disease known as infectious infertility of cattle which has been encountered in Central and Southern Africa during the past few years.

This condition was unknown a generation ago, perhaps because it was unrecognized. But a more likely explanation is that the cattle industries in this area are being rapidly improved with introduced breeds. It seems that the local indigenous native stock is very resistant to it and that the British and Continental breeds are more susceptible. At all events, this disease has emerged within the past ten years as an important factor in sterility in cattle in Africa and, while the knowledge on the disease is in a relatively immature or undeveloped state, authorities there are highly concerned about it and regard it as a grave menace to the cattle industry.

Although information on the disease is limited, the evidence at hand indicates that we must regard the disease as a potential danger and that we should make every effort to prevent its appearance and dissemination here - the least we can do is to recognize it if we should see it.

Definition: This disease is an insiduous and chronic venereal disease of cattle caused by an infectious agent, possibly a virus. It is characterized (1) in cows by a sterility due to a cervico-vaginitis or a metritis and salpingitis, (2) in bulls by a permanent sterility due to an epididymitis and orchitis. Infected animals may remain carriers of the disease for years.

History: The disease was first observed in imported British breeds of cattle in Kenya in 1927. Investigations since then have associated it with sterility in cows due to a cervico-vaginitis or metritis and in bulls with an orchitis. The disease is apparently well known to

the natives of Kenya indicating that it has been present for a long time. Within recent years the disease has been described from most of the Central African and Southern African countries.

Etiology: By a method of elimination it would seem that the causative agent is possibly a virus. No protozoa have been isolated from the affected organs and attempts to cultivate the infective agent on bacteriological media have consistently yielded negative results. However, attempts to filter the agent have also failed but it is thought that this was due to the tenacious consistency of the material being filtered. During attempts to adapt the virus to laboratory animals, several other viruses apparently unconnected with the etiology of the disease, have been isolated in South Africa by McIntosh from the vaginal mucosa of cows, and here McKercher has isolated a virus from the vaginal discharge of cows in California, also apparently quite unconnected with epivag.

The etiology has not been fully determined. It seems likely that certain exciting or predisposing factors are necessary for it to manifest itself.

Transmission: The disease is definitely contagious. It can be conveyed alternately through heifers and bulls by coitus.

The disease can be transmitted artificially with the mucopurulent discharge from the vagina and with emulsions prepared from the epididymis and testicles by the intravaginal method to both heifers and cows and by the intraurethral method to bulls.

The best results at transmission are obtained by introducing a swab soaked in the vaginal exudate or in minced epididymis into the vagina and to leave it there for 24 hours.

In order to obtain good results with transmission, it is important to use English or Continental breeds of cattle - not Zebu or other nondescript types.

Symptoms: The duration of the incubation period varies greatly and depends on the sex, the susceptibility of the animal and possibly also on the virulence of the strain. After a natural infection it varies from 4 to 10 weeks in bulls and usually from 7-10 days, but sometimes from 2-3 weeks in cows. The incubation period following an artificial infection is shorter and varies from 2-4 days in cows and about 4 weeks in bulls.

The disease runs a chronic course which extends over several months or years. Apart from the symptoms and lesions in the genital tract, affected animals do not suffer other symptoms.

A. <u>Symptoms in the bull:</u>

The first symptoms to be noted in the bull are found in the semen. At a very early stage the semen becomes very alkaline and later the sugar content is reduced. Leucocytes appear in large numbers in the semen. The spermatozoa show reduced activity and degenerative changes and eventually disappear completely (azoospermia).

During the early stages of the disease the sexual desire remains normal but the fertility may be lost before there is any clinical evidence of the disease.

The first clinical symptoms are a slight enlargement of the spermatic cord and a puffiness of the epididymis usually on one side. As the disease progresses the enlargement spreads to the head, body and tail and later on the testicle also becomes involved. Affected bulls may still show normal sexual desire despite these lesions.

In advanced cases the testicles appear swollen and knobbly, perhaps one side more than the other, and may hang down lower than usual. On palpation the tail of the epididymis is very hard and enlarged often to the size of a golf ball and sometimes even to that of a baseball. In comparison, the head and body are moderately enlarged. As the disease progresses the testicle undergoes atrophy so that there is eventually an enormously enlarged epididymis and a small testicle. Rectal examination may reveal enlargement of one or both vesiculae seminalis and empullae which are often painful.

The course of infectious infertility in the bull invariably terminates in permanent sterility and impotency.

B. <u>Symptoms in the cow and heifer:</u>

The course of infectious infertility in the cow is varied. The examination of the vagina and cervex are greatly facilitated by the use of a vaginal speculum. Red to reddish purple inflammatory areas or streaks are found on the mucous membrane in the anterior portion of the vagina and cervex several days after infection and about a week later a variable amount (up to 500 ml) of a thick tenacious mucopurulent odorless discharge accumulates in the vagina. Affected cows and heifers may exhibit signs of pain when the speculum is inserted. The excretions are discharged periodically and the vulva, tail and perineal region become

soiled. Microscopically a large number of leucocytes (neutrophils) and moderate bacterial contamination can be detected in the discharge. Apparent recovery takes place after two or three months. Rectal examination may reveal a salpingitis, pavilionitis, peri-ovarian cysts and ovarian cysts.

C. Symptoms in the herd:

The first sign of trouble is noted when cows and heifers fail to hold the service. A few cows frequently returning to the bull suggests that the cows themselves are at fault, but when many or all the cows served by a particular bull return, this obviously indicates that the bull is at fault.

As pointed out earlier, sterility in the bull frequently develops before noticeable lesions in the epididymis can be detected. In view of this, I think it is highly desirable to keep infertile bulls in this country under observation for some months to allow time for the typical lesions to develop which would be one of the best methods of detecting the disease.

The presence of widespread infertility in a herd with profuse vaginal discharge should be viewed with suspicion of this disease.

Pathology: The lesions are confined to the genital organs, but in cows a peritonitis may develop as a complication.

Lesions in the Bull: The lesions are characterized by marked fibriosis - enlargement and distortion of the organs being due to this.

Adhesions are found between the tunica vaginalis propria and tunica vaginalis communis. During the early stages, the ducts of the epididymis are widely distended with a yellow tenacious mucopurulent exudate. On section, this organ shows much fibrous tissue and multiple localized small lacunae containing tenacious yellow fluid. There is a marked proliferation of the connective tissue in the walls of the ducts and in the intertubular tissues and in advanced cases the whole organ consists of fibrous tissue. One is struck by the marked difference in the relative sizes between the hypertrophied epididymis and the atrophied testicle.

At first there is no visible change in the testicle but as the disease progresses the normal yellowish color becomes pale due to the proliferation of the fibrous tissue. Even the walls of the blood vessels of

this organ show evidence of fibrosis. The most important changes in the testicle are due to a proliferation of the fibrous tissue associated with atrophy of the seminiferous tubules and interstitial tissues. The enlargement and induration of the seminal vesicles and ampullae are also due to an increase in fibrous tissue.

Lesions in the Cow: The lesions may involve portion or the whole genital tract. The inflammatory changes in the early stages are visible in the anterior portion of the vagina and a tenacious exudate up to 500 ml is present in the vagina and cervix. These are often no longer visible in long standing cases, when a catarrhal metritis and a small amount of tenacious pus is then visible and the vaginitis has disappeared.

A chronic catarrhal salpingitis has been observed in 15-30 percent of affected females. The Fallopian tubules are contracted at irregular intervals, rather firm and depending upon the duration of the disease a partial or complete stenosis may be present. A pavilionitis may also be present. In a number of cases a local peritonitis in the pelvic cavity has been noted which sometimes may be more generalized.

At times a layer of fibrous tissue may surround the ovary preventing ovulation and favoring the development of ovarian cysts.

Diagnosis: The definite diagnosis of the disease is not easy due to lack of serological and biological tests. A more or less definite diagnosis of infectious infertility can, however, be made in a herd if a case of epididymitis not associated with brucellosis is encountered, though admittedly rarely, there are other causes of epididymitis but these appear in a sporadic form.

In a herd in which the disease has been found every bull with poor semen production, though as yet showing no signs of infection on palpation of the testicles, must be regarded with great suspicion as it may be in the process of developing the disease.

The high prevalence of a cervico-vaginitis and salpingitis in cows is further evidence of the disease.

When a sterility of a serious nature is not explained it may be worthwhile to bear this disease in mind and attempt to exclude or prove its presence. If a bull is sterile, it should not immediately be discarded and slaughtered without further examination. Instead, it should be quarantined and kept under observation for a prolonged period to see if the typical epididymitis develops. In addition, it may be allowed to serve one or more virgin heifers, or alternately emulsions from the epididymis and testicle may be prepared, or vaginal exudate may be introduced into the vaginas of susceptible heifers or cows as described previously.

Differential Diagnosis:

Bulls: The epididymitis must be differentiated from that associated with

(a) Brucellosis
(b) Tuberculosis
(c) Ascending streptococcal infection
(d) Hemorrhagic epididymitis
(e) Corynebacterial infection

Cows: Sterility and infection must be differentiated from infertility or sterility. Following: Trichomoniasis, brucellosis, vibriosis, leptospirosis and various forms of vaginitis, endometritis and salpingitis due to bacterial infections, corynebacteria, strains of streptococci and staphylococci. Faulty feeding and adverse climate conditions may also cause a temporary infertility.

PATHOLOGY OF RINDERPEST

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The object of this paper is to facilitate the recognition of rinderpest by providing further information on the clinical and pathological character of the disease. The information was obtained from the study of a large number of natural and experimental cases. Detailed necropsies were conducted and recorded on over 400 cases in both native African Zebu and European breeds of cattle. Tissues for histopathological study were selected from representative cases. Both kodachrome transparencies and motion pictures were taken to record the lesions on color film and to supplement the written descriptions.

Some 16 strains of the virus were studied. Although these strains were immunologically identical, they varied widely in virulence and hence in the severity of lesions produced.

Mortality varies from 25 to 90 percent in cattle of all ages, depending upon the strain of virus and the resistance of the cattle. The incubation period following experimental administration of the virus is two to three days. Most contact infections require six to nine days.

The typical clinical course of infection with a virulent bovine strain in a susceptible adult bovine animal is briefly as follows: The onset of illness is marked by a sharp rise in temperature to 104 or 105 F., accompanied by restlessness, dryness of the muzzle, and clear tears. Within a day or two of onset, nasal and lacrimal discharges appear; there is photophobia, depression, thirst, starey coat, retarded rumination, anorexia, leukopenia, and excessive salivation. The temperature usually reaches its peak on the third to the fifth day. With the onset of diarrhea, the temperature drops but other symptoms are intensified. Oral lesions may appear by the second or third day of fever but usually do not become conspicuous until after the onset of diarrhea. With increased severity of the diarrhea comes abdominal pain, accelerated respiration, occasional cough, severe dehydration, and emaciation, followed by prostration, subnormal temperature, and death after a course of six to 12 days.

Postmortem Lesions

Rinderpest virus has a high degree of affinity for lymphoid tissue where its effect is degenerative, leading to necrosis of lymphocytes. This is strikingly evident in microscopic sections of the lymph nodes, spleen, and Peyer's patches. Almost complete destruction of lymphocytes in these tissues may take place even though the gross appearance of the

tissue remains relatively normal. Since the capillary endothelium is unaffected, congestion and hemorrhage are conspicuously absent and, in gross specimens, lymph nodes and spleen often appear normal. In severe cases, necrosis in the Peyer's patches may be grossly evidenced by their black color, friable consistency, and tendency to shell out from the mucosa.

Rinderpest virus has a similar affinity for the epithelium of the digestive tract, where it produces early and characteristic lesions. Oral lesions first appear on the inside of the lower lip, on the adjacent gum, on the cheeks near the commissures, and on the ventral surface of the free portion of the tongue. Later they may extend to all areas of the mouth with the singular exception of the anterior seven-eighths of the dorsal surface of the tongue.

Histologically, the oral mucosa is first involved in the depth of the stratified squamous epithelium. Here, just above the basal layer, a small focus of necrosis appears. This necrosis soon extends to the surface and is first seen grossly as a pin-point of grayish white tissue surrounded by normal appearing epithelium. Movement of the tongue and cheeks causes the dead cells to be removed, leaving a sharply walled, deep pit of erosion with a red floor. Later these small pits of erosions widen and coalesce to form larger areas of erosion. The grayish pinpoint foci are difficult to see, hence the erosions are usually the first lesions observed. Similar erosions are found in the pharynx and the initial third of the esophagus. Grossly visible lesions are seldom observed in the rumen, reticulum, or omasum, although erosions of microscopic size may be found. In contrast to the mucosa of the mouth and first three stomachs, the columnar epithelium of the abomasum and intestine is thinner and its vascular bed is closer to the mucosal surface. For these reasons, when the virus invades columnar epithelium, causing necrosis and desquamation of cells, there is a more pronounced vascular response. The capillary bed becomes congested and blood cells readily escape into the stroma. In the early stages, this gives the appearance of petechial or ecchymotic hemorrhages; later, with the loss of larger areas of epithelium, shallow erosions will be recognized grossly.

The abomasum is one of the most common sites for the lesions of rinderpest. The pyloric region is most severely and consistently involved. Necrotic foci of microscopic size in the epithelium, accompanied by capillary congestion and hemorrhage in the underlying lamina propria, result in the gross appearance of irregularly outlined, superficial streaks of color which range from bright red to dark brown. Edema may be profuse in the submucosa of the fundus. As necrosis of the abomasal epithelium progresses, the affected areas assume what is more nearly a slate color and the epithelium sloughs away, leaving sharply outlined, irregularly shaped erosions with red floors from which blood oozes.

Severe lesions are less common in the small intestines than in other regions of the digestive tract. The lesions that do occur vary from streaks and irregular patches of congestion and hemorrhage to erosions and are usually found in the initial portion of the duodenum and terminal part of the ileum. Peyer's patches are often black, friable, and easily peeled out as described previously.

The crests of the folds of mucous membrane throughout the cecum are frequently bright red, due to the presence of many petechiae. Streaks of congestion and extravasation along the crests of the folds of mucosa give it a characteristic barred or so-called "zebra-striped" appearance. Erosions and hemorrhage may occur. The mucosa in and adjacent to the ileocecal value is frequently involved, but the most severe changes are seen at the cecocolic junction apparently because of the presence of lymphoid tissue, diverticula of the mucosa, and susceptible epithelium. Congestion, hemorrhage, and erosion of the mucosa and edematous thickening of the intestinal wall are frequent in this area.

The lesions in the colon and rectum are similar to those which appear in the cecum, the terminal portions of the rectum being most commonly involved.

The liver occasionally shows passive congestion but no specific lesions. Lesions in the gallbladder are similar to those in the lower part of the intestinal tract. They vary from scattered petechiae to diffuse blotches of hemorrhage throughout the mucosa. The gallbladder is usually distended with bile.

In the respiratory tract, the turbinates often present petechiae. Lesions in the larnyx vary from a few petechiae to small areas of mucosal erosion. In the anterior third of the trachea, longitudinal streaks of rusty red hemorrhages are typically found. These are associated with varying degrees of mucosal engorgement. Erosions are uncommon. Lungs appear to be only secondarily involved. Emphysema, both interlobular and alveolar, is the most common lesion in the long-standing, severe cases.

The lesions in the heart are usually limited to diffuse subendocardial hemorrhages in the left ventricle near the papillary muscles.

Lesions in the kidneys are usually confined to congestion, particularly at the corticomedullary junction. The mucosa of the urinary bladder may show varying degrees of congestion or hemorrhage.

<u>Summary</u>

Several hundred experimental cases of rinderpest in cattle, produced by a variety of virus strains, were studied clinically and at necropsy. Histopathological changes were found which help provide a more complete understanding of the clinical and gross manifestations of the disease.

Rinderpest virus has a selective affinity for both lymphocytes and epithelium of the mouth and digestive tract. In the stratified squamous epithelium of the mouth and esophagus, it produces necrosis and erosion of epithelial cells of the malpighian layer with relatively little change in the underlying vessels. In contrast, the columnar epithelium of the abomasum and intestine, and the proximity of its highly vascular stroma, results in relatively severe congestion and hemorrhage associated with necrosis of the epithelium. Congestion, hemorrhage, and occasionally erosions also occur in the trachea, urinary bladder, and gallbladder. The Peyer's patches and mucosa of the cecocolic junction, which have an abundance of lymphoid tissue, are particularly affected by the virus. The lymphocytes in the lymph nodes and spleen are almost completely destroyed.

Subendocardial hemorrhages in the left ventricle, pulmonary emphysema, and passive congestion in the liver and kidneys occur secondarily but are helpful in diagnosis.

The clinical course and lesions produced by rinderpest are characteristic and presumptively diagnostic.

COMPARISON OF MUCOSAL DISEASES AND RHINOTRACHEITIS OF CATTLE

1956

	MUCOSAL DISEASES				RESPIRATORY DISEASE
Reference	Rinderpest[1]	Mucosal Disease[2]	Indiana V. D.[3]	New York V. D.[4]	Rhinotracheitis[5]
Incubation Exp.	3-5 days	2-8 days	2-5 days	3-7 days	3-5 days
Temperature	Above 104	2-3 days above 104	Above 104	Above 104	105-108
Leukopenia	Severe	Yes	Yes	Yes	No
Rhinitis	Yes	Occasionally	Yes	Yes	Yes
Trachea Inflammation	No, localized hem.	No	Yes	No	Yes, severe, exudate
erosions	Yes, anterior 1/3	No	No	No	No
Lacrimation	Yes	Inconstant	No	No	Inconstant
Corneal Opacity	No	Yes, 10%	Rare, calves	No	No
Diarrhea	Yes	Yes	Yes	Yes	Occasionally
Buccal Erosions	Yes	Yes	Yes	Yes	No
Erosions on Dorsum Tongue	No	Yes	No	No	No
Infl. Hem. Erosion Digestive Tract	Yes	Yes	Yes	Yes	No
Lymphoid Tissue Damage	Yes, severe	Yes	Yes, mild	Yes ?	Edema, Hem.
Lameness	No	Yes, 10%	Yes, 10%	No	No
Morbidity, Herd	100%	2-50%	80-100%	33-88%	to 100%
Mortality, Case	90-100% U. S.	90%	0-50%	4-50%	3%
Transmissible Agent	Yes	Yes	Yes	Yes	Yes
Agent Isolated	Yes	No	Yes	Yes	Yes
Immune Animals	Yes	Yes	4 months	Yes	Yes
Immune Serum	Yes	?	Yes	?	Yes
Neutraliz. Antibody	Yes	?	?	?	?

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AFRICAN SWINE FEVER

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African swine fever is an acute, febrile, highly contagious, virus disease of swine. It is characterized by an acute course, a very high mortality, and gross lesions which closely resemble acute hog cholera. An inapparent transmissible infection is produced in wart hogs and other wild swine which serve as carriers. No other species have been found susceptible. The virus producing the disease is immunologically distinct from that causing hog cholera, and animals immune to hog cholera are fully susceptible to African swine fever.

Montgomery[1] reported East African swine fever as having first been seen in East Africa around 1910. Steyn's[2] description of swine fever in South Africa first pointed out the similarity to East African swine fever. It appears from these early reports that by 1926, so-called East African swine fever was present in both South Africa and East Africa. The disease has persisted in both of these areas to date. The disease also occurs in West Equatorial Africa where in some areas it prohibits the commercial raising of swine. The severity of some of the early outbreaks in South Africa is indicated by the report of DeKock.[3] He described 11,000 animals involved in an outbreak in 1933 and 1934. Of these, 8,000 died, over 2,000 were slaughtered in an emergency slaughter program, and only 862 were considered survivors.

The disease is caused by a filterable virus which is present in the blood, tissue fluids, internal organs and all excretions and secretions of infected animals. Under African field conditions the African swine fever virus is carried by wart hogs (Phacochoerus), giant forest hogs (Hylochoerus) and bush pigs (Potamochoerus), in which it produces an inapparent infection. Contact between infected wild porcines and the domestic breeds of swine initiate many of the outbreaks in Africa. Once established in domestic swine the infection spreads rapidly by direct contact or through contaminated feed. Some field experience suggests that an insect vector may be involved in transmission but experimental evidence is lacking. The virus is a very stabile one. DeKock[3] reports survival of the virus in blood stored in a cold dark room, for a period of 6 years. Virus in blood will survive several weeks at room temperature and contaminated pens are infectious for at least 2 weeks in the tropics.

Inasmuch as there have been very few survivors, immune serum has not been available for study. Therefore very little, well established information has been obtained on the immunity produced by the African swine fever virus or of the antigenic character of different strains.

The unsuccessful attempts by Walker[4] and others to produce a vaccine, in part led them to believe that the virus was capable of changing its antigenic character after a few animal passages. Further, Walker's experience indicated that recovered animals were only immune against the homologous virus which produced the infection. Recovered animals have carried the virus for at least 10 months. When convalescent sera obtained from such recovered animals is heated and injected in large volume simultaneously with a minimum infective dose of African swine fever virus, test swine seldom survive. Such convalescent sera have demonstrated a very low protective titer even against homologous virus. No serologic test is available for diagnosis and no vaccine has been developed.

Following a natural incubation period of from 5 to 9 days, the temperature rises abruptly to above 105.0° F. where it stays for about a four-day period. Distinct clinical symptoms are usually apparent only during the 48-hour period of declining temperature prior to death. The relationship of the clinical features of this disease to the temperature curve is unusual and in contrast with that in hog cholera. The most distinctive symptoms are depression, weakness, anorexia, cyanosis of the skin, cough and dyspnea. Diarrhea and vomiting may occur with some strains of virus. Death usually occurs by about the seventh day of fever. The mortality rate invariably exceeds 95 percent and usually approaches 100 percent. Most recoveries are reported to have occurred among those animals which have been infected by contact with wild pigs; establishment of the virus in domestic swine so enhances its virulence that recoveries are rare. The few survivors carry the virus for many months.

Pathology: Cyanosis of the ears, snout, abdominal wall and legs are often striking in light colored pigs. More discrete hemorrhages are also seen, particularly on the legs and abdominal wall. These hemorrhages usually have dark centers and fading edges.

The larynx, particularly the epiglottis, usually bears petechial or ecchymotic hemorrhages. In some instances it is deeply congested and presents more severe hemorrhages than occurs in hog cholera.

An excess of straw-colored pleural fluid is frequently present in the thoracic cavity. A variable number of petechial and ecchymotic hemorrhages occur on the serous surfaces and in the parenchyma of the lungs in nearly every case. Edema, often marked by broadened edematous interlobular septa is of frequent occurrence. Small areas of atelectasis in the cardiac lobe of the lungs is a very common occurrence, but in most instances appears to antedate the African swine fever.

There is often an excess of pericardial fluid. Some degree of cardial hemorrhage is observed in about 70 percent of the cases. Subepicardial and endocardial hemorrhages are the most frequent occurring and may be diffuse, very extensive and striking in appearance. They often appear to emanate from the coronary vessels and are most marked over the left ventricle. In rare instances there may be some edema in the areas involved by hemorrhages. Subendocardial hemorrhages are also most conspicuous in the left ventricle. Grossly the myocardium is usually normal except for the hemorrhages.

The lymph nodes present the most constant and striking lesions, being edematous and extremely hemorrhagic. The visceral nodes are usually hemorrhagic to a degree rarely if ever seen in hog cholera, appearing superficially and on cross section more like hematomas than nodes. The nodes in the musculature usually have peripheral hemorrhage grossly indistinguishable from those in hog cholera.

About half of the spleens examined grossly appear normal. Petechial hemorrhages, congestion with enlargement and small areas of infarction occur. Lesions are more consistently found histologically than grossly.

The liver appears grossly normal in over half of the cases. The most consistent lesion is a mottling with areas of congestion. Small hemorrhagic areas are also of occasional occurrence. Yellow areas, grossly indicative of fatty changes are seen in about 10 percent of the livers. Another occasional change is the engorgement of blood vessels and edema in the hilus adjacent to the gallbladder. The most common lesion seen in the gallbladder is the engorgement of blood vessels which is inconspicuous in cholera. Petechial or ecchymotic hemorrhages may be scattered over the mucosal and serosal surfaces. The gallbladder is usually distended with bile.

The most constant lesion in the kidney is hemorrhage which occurs in about two-thirds of the cases. Petechial hemorrhages, most numerous beneath the capsule, also appear in the cortex and pelvis. In a few cases, severe, diffuse hemorrhages occur in the pelvis, subcapsular region and in the perirenal fascia. Edema often accompanies the diffuse hemorrhage.

The most common lesion in the stomach is an acute, diffuse, often hemorrhagic gastritis which is most severe in the fundus. The hemorrhages vary from petechia to diffuse with free bleeding. Ulcers, often covered with necrotic debris, appear in the pyloric and fundic region in about one-fourth of the cases.

The small intestines present varying degrees of inflammation or hemorrhage in about two-thirds of the cases. The enteritis varies from localized, mild, red areas, with petechial hemorrhages on the crest of the mucosal folds to generalized severe inflammation and diffuse hemorrhage. Erosions are not observed. Scattered subserosal petechia and ecchymoses may occur. In a few cases the Peyer's patches are visible on the serosal surface as yellowish, edematous areas sprinkled with petechia. The mesenteric blood vessels of the small intestine are consistently engorged and very striking in appearance.

The ileocecal valve presents lesions in about one-third of the cases. The lesions vary from mild inflammation to hemorrhage, with ulceration and adherent necrotic debris.

The cecum shows significant changes in about 50 percent of the cases. The lesions vary from mild reddening to severe hemorrhage with ulceration of the mucosa. Petechia, along with blotches and longitudinal streaks of diffuse hemorrhage are most common. When ulcers develop, they are usually small, deep, and covered with necrotic debris. Lesions simulating the "button ulcers" of hog cholera are unusual and occur in the cecum or colon only in the more long standing cases. Subserosal hemorrhages are present in a few cases.

Inflammation of the colon occurs in about 50 percent of the cases. It varies in severity from a reddening through petechiation to an extensive confluence of ecchymosis. Small ulcers, such as seen in the cecum may be present. In some cases hemorrhages occur deep within the wall, which are equally evident from either the mucous or serous surface. Hemorrhages may also be present on the serous surface with edema in the mesentery.

It is apparent from the foregoing description that the clinical features and gross lesions of African swine fever are so similar to those of hog cholera that careful observation, examination and necropsy of several animals from a suspect herd is required to aid in differentiation. It must also be noted that some individual cases of either hog cholera or African swine fever may present only very meager inconspicuous lesions. In such individual cases, it may not be possible to differentiate the two diseases by the clinical features and gross lesions. Differentiation is best made on a herd basis and must always be supported by animal inoculation.

African swine fever should be suspected when pigs immune to hog cholera develop a cholera-like disease with the following features:

First: - a rapidly fatal, highly contagious, febrile disease in swine which frequently fail to present clinical signs until within 48 hours of death.

Second: - gross lesions similar in character, but more severe than those of hog cholera. This applied especially to: cyanosis and hemorrhages of the skin, hemorrhages of the lymph nodes, hemorrhages on the heart and kidneys, pulmonary interlobular edema, congestion and edema of the gallbladder and generally more severe vascular damage.

If these conditions are found, the outbreak should be reported to State and Federal animal disease control authorities for a confirmatory diagnosis. A definitive diagnosis can be accomplished by histologic and immunologic means, which includes the inoculation of swine known to be immune to hog cholera.

Prevention and control are presently dependent upon an awareness of the ever present threat of African swine fever, import restrictions, prompt diagnosis, quarantine and slaughter. The need for research work on this disease is obvious and urgent.

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TESCHEN DISEASE

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Teschen disease is an acute febrile contagious virus disease of swine affecting the central nervous system.

The disease was first described in 1929, by Trefny, in the town of Teschen, Czechoslovakia. It became widespread during World War II, and is still present and important in Czechoslovakia and other central and western European countries where it has often reached epizootic proportions causing severe losses. There were some 50,000 cases in 1944, in Czechoslovakia where Teschen disease is considered the next most important disease of swine after erysipelas and hog cholera. The disease has been reported only in Europe and Madagascar. It has not been recognized in the Western Hemisphere.

The incubation period varies from six days for experimental transmission to three or four weeks following field exposure. From 70 to over 90 percent of affected pigs die. Domestic and wild swine are the only susceptible species. Laboratory animals are not susceptible even to intracerebral inoculation.

The virus is filterable, quite resistant to drying, and retain infectivity for over a year when frozen or in 50 percent buffered glycerine at 0^{o} C. The similarity of some of the lesions in the central nervous system in Teschen disease to those occurring in human poliomyelitis had suggested the possibility of a relationship between the two diseases but the virus of Teschen disease has proven immunologically distinct.[1] The presence of virus in tissues outside of the central nervous system is variable and transient as indicated by the usual failure of attempts to recover it from tissue, blood or discharges. The virus is most consistently found in tissues of the central nervous system where it is at the highest concentration when the animal is paralyzed and the temperature is at its peak. The brain and cord are best pooled as a source of inoculum for experimental transmission. Young pigs are most susceptible and intracerebral inoculation results in over 80 percent infection with clinical symptoms. Results of experimental transmission by intranasal or intramuscular routes have been inconsistent. Intraperitoneal, subcutaneous, intradermal and intravenous routes of virus administration usually fail to produce the disease. Some swine have been infected by feeding with infected central nervous system tissue. Natural transmission is accomplished primarily by direct contact. Infection by means of feed contaminated with the discharges of infected animals is considered a possibility but experimental evidence is lacking.

Insects are very unlikely to be involved in transmission because the virus is seldom found in the blood and animals are not usually susceptible to subcutaneous inoculation. Furthermore, the disease occurs in all months of the year, regardless of the presence of insects.

Clinical onset is marked by a moderate temperature rise to a possible 106° F. Included are lassitude, depression, conjunctivitis, and sometimes incoordination of the rear legs. There follows, after one to three days, a period of central nervous system irritability with stiffness, falling, tremors, nystagmus, and violent clonic convulsions, possibly with squealing which may be prompted by a sudden noise or handling. Stiffness and opisthotonos may be the most pronounced symptoms. Animals frequently lie on their side with running motions or may sit on their hindquarters in a dog-like manner. The appetite usually remains good and the animals will eat and drink eagerly if held up to the feed. The temperature is only moderately elevated during this period. Paralysis usually starting with the hindquarters may slowly involve all 4 legs and the entire body. Convulsions may occur during the paralytic stage and the voice often becomes altered. Death usually results from respiratory paralysis. Residual paralysis is common in recovered animals.

Pathologic changes: The few nonspecific gross lesions which may occur are of secondary nature. The only specific lesions are the microscopic ones associated with degenerative changes, in the brain and spinal cord.

In the spinal cord the most striking, constant and characteristic change is limited to the gray matter, particularly the neurons in the ventral horn. These cells may show all stages of degeneration; chromatolysis, vacuole formation, karyorrhexis or lysis and neuronophagia. There is perivascular infiltration primarily of lymphocytes and plasma cells. Neutrophiles are rarely involved. Both focal and diffuse glial proliferation occurs. These changes are most pronounced in the cervical and lumbar cord areas.

Lesions in the brain are similar to those in the spinal cord. In the cerebellum, degenerative changes occur in the granular and molecular layers and in the Purkinje cells. Lesions are also of frequent occurrence in the thalamus. The pons and medulla are least affected. Although the motor cortex of the cerebrum is involved it is not the primary seat of lesions as in primates with poliomyelitis. The meninges over the cerebellum may be infiltrated with lymphocytes.

Teschen disease should be suspected when a febrile disease of pigs occurs with central nervous system symptoms of irritability, tremors, convulsions and flaccid paralysis starting in the hind legs. The diagnosis can be confirmed by histologic study of the lesions in the spinal cord and brain as noted above and by recovery of the virus. Virus isolation is most readily accomplished by the intracerebral inoculation of

susceptible young pigs with pooled central nervous system tissue from the cerebellum, cord and cerebrum of the infected animals. When a transmissible agent is obtained, swine immune to hog cholera, should be inoculated.

Formalinized and phenolized brain and spinal cord tissue vaccines have been used with only partial success. Traub[2] has prepared an aluminum hydroxide absorbed formalized brain tissue vaccine which is an improvement over earlier ones.

Prompt recognition and diagnosis are essential. The appearance of the disease in new countries should be followed by prompt efforts to eradicate the disease by quarantine and slaughter.

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THE VESICULAR DISEASES

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The vesicular diseases of animals -- foot-and-mouth disease (FMD), vesicular stomatitis (VS), and vesicular exanthema (VE) -- can be described as acute infectious diseases of animals, characterized by fever and by the formation of vesicles on the mucous membranes in the mouth and on the skin, especially of the feet, dew claws, teats, and udder. Although this description fits all three infections, even the most experienced specialist in his field is unable to differentiate between them without some diagnostic aids or laboratory assistance. Moreover, it is extremely important that the veterinarian recognize the existence of a vesicular disease if it occurs and immediately report its occurrence to the proper office.

Etiological Agents: All three of the vesicular diseases are caused by filterable viruses, but although all three infections are clinically indistinguishable, the agents causing the respective diseases differ in particle size. The FMD virus was found to be 8-12 mu, VS virus 60-100 mu, and VE 14-22 mu.[1,2,3]

Plurality of Viruses: Early investigations regarding immunity to FMD indicated that the duration between outbreaks in the same animals varied considerably. Some investigations reported that cattle were reinfected in 6 to 10 weeks; in other instances reports indicated that reinfections occurred 10 days following recovery from the disease.

Investigations conducted in many European institutions eventually showed that there was more than one immunological type of the virus. In 1922, two French workers, Valle and Carre, reported the existence of two types of FMD virus.[4] Each type produced a typical form of the disease clinically indistinguishable from the other. Upon recovery, however, animals were immunized against the homologue but not against the other. They designated one type as A (Allemand) and the other as type O (Oise). Four years later Waldman and Trautwein announced that they had found three immunologically distinct types of the virus in Germany and designated them as types A, B, and C.[5] These findings were amply confirmed, but the German type A and B corresponded with the French type O and A respectively. To avoid confusion, it was generally agreed to retain the Frenchmen's designation.

Symptomatology[6,7,8,9,10]: The virus of FMD, VS, or VE, when introduced naturally or experimentally at some susceptible sites, such as the mucosa of the mouth or epithelium on the tongue, results in an invasion that produces a typical vesicle. Artificially, vesicles develop in about 24 hours or less; however, in some cases, because of a low virus content in the infectious material, or because of some strains of the virus, there might be some delay from 48 to 60 hours. Under field conditions the incubation period in natural infections varies from 2 to 7 days. Occasionally it may be longer. Prior to the formation of the vesicle there may be a rise in temperature, which in the young, robust animal is well marked. According to Hutera and Marek, older animals with FMD sometimes show a very slight increase in temperatures which may escape notice. With VS it has been observed that old cattle appear to be more susceptible than younger animals.

The rise in body temperature is believed to be due to the entrance of the virus into the blood stream. This is soon followed by the formation of the primary vesicle. The vesicles usually occur on the tongue or the mucous membranes of the mouth; before their formation the lips and gums may become hot, dry, and reddened. When the mouth is opened, quantities of accumulated saliva may escape, giving the animal the characteristic picture frequently seen in textbooks. The lesions in the mouth may be small in mild infections or very extensive in severe cases. The mucous membranes involved may be the inner surfaces of the lips and the opposing membranes of the gums, or they may be those on the dental pad, on the dorsum of the tongue, on the frenum, and less frequently on the cheeks. It is also important to examine the mucous membranes at the angle of the jaw. These vesicles, before rupturing, are thick-walled, giving the surface an uneven or undulating appearance. In other instances, they may be thin-walled and almost transparent containing a clear, colorless or slightly yellowish fluid that later may become turbid and greyish-white. After one to three days the vesicles rupture and expose a moist red or raw, hemorrhagic, painful erosion, surrounded by a white-to-grey margin of ragged fragments representing the remnants of the vesicle. The erosions in several days become covered with fresh epithelium which eventually disappears without leaving a trace of the lesions. As soon as the lesions are healed, the animal may resume eating.

Occasionally, vesicles may develop on the nasal mucosae and the conjuncitiae. The process may extend to the pharyngeal mucosa which may give rise to symptoms of a pharyngitis. Sometimes in cattle, the cornea may become cloudy and may present small vesicles. Occasionally vesicles develop on the epiglottis and in the mucosa of the trachea and the bronchi, causing symptoms of dyspnea and pulmonary edema. Lesions found in the rumen have also been reported.

In a typical biphasic course of the disease, the virus in the blood stream reaches the sites of predilection, producing secondary vesicles. The formation of secondary vesiculations may or may not be preceded by a second rise in body temperature. Infection of the hooves (secondary) may develop simultaneously with infection of the buccal mucosa (primary) or may be delayed from one to several days. Not in all cases will the biphasic reaction be observed. Animals naturally or artificially infected with VS more or less frequently develop secondary lesions, which are less frequent with FMD and VE. The formation of vesicles at the secondary sites is influenced to a great extent by pressure in those areas. Therefore, secondary lesions may be more frequently observed in heavy animals, in inoculated animals allowed to walk on rough ground, and in the laboratory when the experimental animals are allowed to walk on wire cage floors.

In the field under natural conditions the vesicles may be observed as primary or secondary, and the point of entry may not be readily observed. For example, the infection may have been introduced through the digestive tract, establishing the primary vesiculation in these organs, such as the posterior portion of the mouth or pharnyx, or even in the rumen in the case of FMD. The teats and udder lesions may also serve as primary sites as a result of milking or nursing by the infected young, or they may be secondary lesions following a primary lesion in some other parts of the body.

The first symptom when the hooves are involved is pain, which causes lameness or stiffness of gait. If the lesions are found on more than one foot, the animal shows such pain that it lies down and has difficulty in standing. On examination, the skin around the coronary band, the pad, the cleft of the hoof is hot, swollen and painful and the unpigmented tissues may be slightly reddened. After one or two days, small vesicles may appear which may attain the size of a hazelnut or even larger and contain a clear to turbid fluid. These may rupture and a brown crust may be formed; removal of the crust exposed the moist, red, sensitive erosion. New epithelium slowly forms under the crusts, and the pain and swelling subside in about one or two weeks.

On the udder the vesicles develop on the teats and may sometimes reach the size of a nut. The entire udder may become swollen and the skin around the vesicle is reddened and painful. The vesicles usually rupture on the first day during milking; if protected from mechanical injury, they may persist for as long as 5 to 6 days before rupturing. After erupting, these erosions usually heal under crusts.

Vesicles may also develop in other parts of the body. Occasionally the mucous membranes of the vulva and the vagina may be involved and more rarely on the skin of the inner side of the thigh and on the chest

or the abdomen. These eruptions are of less importance. More frequent and of greater importance is the development of vesicles in the pharynx, larynx, trachea, and bronchi, which, by inhalation of their contents, may give rise to a bronchial pneumonia.

Complications frequently occur during the course of the disease. Secondary infection may take place after rupture of vesicles by the invasion of pyogenic bacteria. Other complications are abortion, septicemias, and inflammation of the hooves which causes the horns to separate from the corium and may even result in the loss of the hoof.

Malignant Foot-and-Mouth Disease: A malignant form of FMD has been described which is fatal in the absence of complications because the virus directly affects the heart muscle. The symptoms are much more severe. The causes for the appearance of this form of the disease are not completely known; it probably results from the degree of virulence of the virus and susceptibility of the animals. This form of the disease has most frequently been found following the great wars, when the disease spreads rapidly among animals. The mortality in most FMD outbreaks is relatively low, but in the malignant form it may be as high as 50 percent, particularly in the younger animals in a herd.

Foot-and-Mouth in Other Animals: The symptoms in sheep are, as a rule, milder and may be so mild as to remain unrecognized. This is especially true in cases where the disease is limited to the buccal mucosa and when the vesicles are small and heal very rapidly. On close examination in such cases, swelling of the upper lip and sometimes of the cheeks and pharyngeal regions may be seen. Lesions on the feet in such cases are similar to those in cattle, except that the vesicles are smaller and heal more quickly. Lameness and avoidance of unnecessary movements may be the only clinical manifestations of foot involvement.

In goats the disease is often mild and presents symptoms similar to those in sheep. Occasionally, severe forms occur where the losses may reach to 10 to 30 percent or higher. The feet are rarely and only mildly affected. In more severe infections the general symptoms, such as high temperature, lassitude, and decrease or loss of appetite, may appear. Suppression of milk is pronounced from the very beginning, and swellings of the head, chiefly of the cheeks and lips are often observed.

In swine the course of the disease does not materially differ from the description given for cattle. The disease principally affects the feet and more rarely the buccal mucosa and snout; it may be recognized in most cases by lameness.

Course of the Disease: In a great majority of cases the course of the vesicular disease is favorable. The vesicles usually heal within 5 to 6 days, but even in the benign cases healing may take 2 to 3 weeks, depending upon the parts of the body affected. Affections of the mucous membranes heal within a week, whereas those on the feet are of longer duration, especially if the erosions are infected by pyogenic bacteria. After the lesions have healed the animals soon regain their normal condition. In unfavorable conditions, excluding the malignant form of FMD death is usually due to a septicemia resulting from a secondary pyogenic bacterial infection or to a gangrenous pneumonia.

Strains of Virus and Virulence: Variations in virulence of the FMD virus outbreaks have been noted in Europe in the past. The great epidemic that began in Germany in 1888 steadily increased until 1892 when it diminished gradually for a few years, but the disease reappeared and reached great proportions in 1899. Thereafter, it continued until 1911 when it reached a virulence previously unequaled. Approximately 1 of 7 susceptible animals was infected. The government attempted to stop the epizootic by the slaughter of affected herds, but the disease had gained too much headway and was too firmly established for this method to succeed. The common practice, therefore, was to expose susceptible stock to mild cases in the hope that they would develop a mild attack which would confer an immunity.

Another severe outbreak occurred in Europe in 1919 through 1921, after World War I, in which the high virulence was attributed to the rapid passage of the virus from animal to animal, inasmuch as the population had been depleted during the war and was replaced with young and highly susceptible individuals.

In VE outbreaks we have observed certain strains of the virus which upon inoculation into susceptible hogs produced very mild forms of the infection. Typical vesicles did not always develop following inoculation. The lesions were small and did not form a typically raised, blanched epithelial vesicle but appeared granular when observations were made at 24- and 48-hour intervals. The typical vesicle may have occurred, although the stage, as often described, was transitory. Fever was not always observed in the inoculated animals, and the incubation period with this strain was prolonged to 48 or 72 hours. Secondary lesions have been observed only on rare occasions in hogs that had been inoculated with this virus four months previously. This would indicate that pathogenicity of VE is not only dependent upon the host but is also associated with the character or strain of virus involved.[19]

A study of the outbreaks of VE in California during the past five years reveals that the severity of the disease in a herd is not entirely dependent on the immuno-type involved, but rather on the strain type of virus.[11]

In a study of the epizootiology of VE, a group of 60 susceptible hogs was introduced onto an infected premise; inspection of the individual hogs at 5- to 8-day intervals revealed that the herd suffered two distinct outbreaks of the disease. Inspection of the experimental herd on the sixth day showed that 16 hogs, or 32 percent, presented very mild but typical vesicular lesions, caused by a virus designated as No. 330 (type F)*. On the 35th day of observation and 40 days after arrival of the hogs, another outbreak of VE occurred, which cross-immunity tests showed to be immunologically distinct from the first.** Typical lesions were observed in 47.1 percent of the hogs. In both outbreaks the disease was considered extremely mild. There was no marked effect on appetite. The animals appeared alert and did not present symptoms of lameness. Although only a relatively small percentage of hogs showed clinical evidence of the disease, 90 to 100 percent of the hogs were infected. Hogs not visibly sick suffered from subclinical infection as subsequently determined by challenge with the homologous strain of virus.

In another group of 20 hogs observed daily, only one showed a small vesicle in the snout during the 20-day observation period. On challenge, however, all of the hogs tested were immune to the homologous virus three weeks later. These data definitely demonstrated the existence of subclinical infections with VE virus under natural conditions. Subclinical infections are also reported for FMD. Gallaway, Brooksby, and Henderson made a careful comparative study of the strain of virus isolated from the recent outbreak that occurred in Mexico, and found it to produce a considerably higher rate of inapparent infections in cattle than the same immunological types isolated in England.[47]

This diversity of the characteristics of virus strains should be particularly stressed to inspecting veterinarians to impress upon them that the vesicular diseases do not always manifest themselves typically or clinically in 100 percent of the animals as so frequently illustrated and described in the texts.

Adaptability of the Viruses: In war between men the element of surprise is the aim of the generals. In our war with infectious agents the latter use the same principle in attempts to defeat us. Once we become sufficiently complacent with our present program of prevention, we may expect a surprise. We, therefore, must always accept the possibility that any of these viruses may lose infectivity for certain species.[12,13,14,15] As an example, the virus of FMD in the outbreak in Germany in 1927 and 1928 presented a strong and peculiar adaptation for hogs. In these cases, the disease was readily transmitted to

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* 6th immunologically distinct type (to be Published).
** Type unidentified at time of writing.

guinea pigs but was not pathogenic for cattle. Unfortunately, no mention of tests on horses was made. In the 1920 outbreak in the same country, a predilection of FMD virus for sheep and goats was observed. The virus harvested from these species was only slightly infective for cattle and swine. This finding was contrary to the usual observation that cattle and swine are more susceptible than sheep and goats to the FMD virus.

As another example of adaptation, the British FMD research committee reported several instances in which the virus of FMD obtained from Argentina produced lesions in cattle but not in guinea pigs. German researchers, also working with a virus of FMD isolated from cattle in Argentina, showed that the agent could produce an excellent disease in swine and cattle, but they were unable to induce the disease in guinea pigs throughout half a year of experimentation. In spite of these experiences, many of us may be laboring under the wrong impression in thinking that all strains of the vesicular viruses must produce lesions in certain species of animals and that failure to do so is an indication that the virus has perished. Actually, the failures are due to a strong adaptation of the virus from which it was originally secured and to its lack or loss of infectivity for another species.

Histopathology: Histologically the vesicles produced by all 3 viruses consist of a circular area eaten out of the stratum malpighii.[7,16] The center of the area usually contains only cellular debris and fluid. In extremely early stages the cells lining the area usually display a cytoplasmic degeneration with pyknotic nuclei and are surrounded by cells showing typical ballooning of the cytoplasm (hydropic degeneration). There is a marked stretching of the intra-cellular tissue and considerable intercellular edema. A few normal epithelial cells may be found around the region of the edema, but usually one vesicle blends in with another. The subcutaneous connective tissue shows an acute inflammatory change characterized by congestion, edema, hemorrhage, and a polymorphynuclear infiltration. No inclusion bodies have been described for any of the three vesicular diseases.

Reports of some FMD outbreaks involving the malignant form of the disease describe a parenchymatous degeneration of the myocardium and necrosis of the muscle tissue, which is manifested by small, discrete or confluent, gray, white, or yellowish areas usually described as a "tiger heart". Unweaned mice and day-old chicks inoculated with an adapted virus of FMD show that these muscles are definitely affected.[17]

Immunization: The immunity produced following either a natural or artificially induced FMD[18] may be one of two types -- the local

(histogenic) or the general (humoral). In FMD the local immunity develops within three days about the site of inoculation, whereas the humoral immunity is detected within a week but requires 3 to 4 weeks to reach its peak. The local immunity to contact infection with the virus usually lasts 3 to 4 months, so that six months after an animal has recovered from an infection, inoculation on the tongue may produce a typical vesicular formation at the site of inoculation with no marked systemic reaction. The humoral immunity against a disease, however, may persist for two to three years; however, this immunity usually begins to wane after a year's time.

Vaccines against FMD, the best of which are produced by absorbing the virus on aluminum hydroxide and inactivating it with formalin are effective for about four months. Such vaccines have been used in Europe and in the Mexican outbreak.

Hyper-immune sera, as well as blood serum taken from animals that have recovered from the disease, confer a passive immunity that may last 4 to 6 weeks, long enough to give some protection to cattle in enzootic areas. Hyper-immune sera are used more extensively in Europe and in the South American countries.

Like FMD, VE apparently produces a good local, as well as a general, immunity following a natural or artificially induced infection. The local immunity appears to be more substantial and longer lasting than that occurring after FMD; in fact, hogs that have recovered from an artificially induced type C infection were found to be immune to inoculation with the homologous virus for at least 710 days.[19] No vaccine is commercially available for VE.

Cattle that have recovered from an infection of VS virus develop an immunity of short duration. Within 30 to 60 days after recovery, many animals can be reinfected experimentally to produce the clinical form of the disease, yet these animals may possess significant neutralizing and complement-fixation antibodies. In field cases, the circulating antibodies usually disappear from the herd within six months.[9,20] It is apparent that carriers must be extremely rare or enzootic foci would be more common.

Diagnosis: The typical vesicle, showing the characteristic blanching and lifting from the normal tissues below and containing the clear-to-colorless or straw-colored fluid, is pathognomonic of the vesicular diseases. However, a differential diagnosis should be conducted by veterinarians trained in the diagnostic procedures. Such men are readily available in all parts of the United States and are usually attached to state or Federal agencies. The diagnosis of the vesicular

diseases is made by two general methods: (1) in the field by the inoculation of animals, and (2) in the laboratory by serological methods and by the inoculation of laboratory animals.

Species Susceptibility: Under field conditions a diagnosis can be made by inoculating several species of animals to determine the host range of the virus suspected. Inoculation of the suspected material into horses, cattle, and swine resulting in infection in all three species indicates a VS infection. The susceptibility of swine and cattle, but not horses, suggests FMD. Susceptibility of only swine to the material indicates VE.[8,15]

The horse may at times react to an inoculation of VE, but the lesions are very mild, small, well-circumscribed, and blanched but not typically raised. Removal of the necrotic epithelium presents a well-demarcated erosion leaving a typical crater with a moist, red, and painful base.

Gallaway and Hove[21,22] found both cats and dogs susceptible to inoculation with FMD and reported the disease to be transmitted by contact from one animal to the other of the same species. Hutera and Marek[10] also refer to the susceptibility of dogs to FMD; however, a number of workers were unable to transmit the infection experimentally.[23]

Dogs with lesions on the tongue in epizootic areas of VS have been reported.[24] Experimentally, however, a number of workers were unable to reproduce the disease.[20]

Experiments with VE in the dog showed that the tongue epithelium was irregularly susceptible to invasion by three immunologically distinct types of the virus (A, B, and C).[25] Dr. J. Julian of California reported lameness with swelling of the pads in a shepherd dog used as a working animal in a herd of infected hogs. The lesions, upon inspection, were indistinguishable from the lesions in hogs. The vesicles occurred around the toes and between the claws, with loosening of the pads. Unfortunately, no serological or virus isolations were made on the dog in the VE outbreak or on those observed in the VS outbreak.

Diagnosis by Laboratory Methods: The vesicular diseases can be diagnosed by the complement-fixation test. This test is used for identification when the vesicular coverings and fluids from a suspected case are submitted to the laboratory. The harvested material should be fresh, placed in a phosphate-buffered glycerin solution (pH 7.4), and kept refrigerated or frozen. This material is used as the antigen and tested in the laboratory against specific hyper-immune sera of the various vesicular diseases.[26]

The test has been found to be successful but has some drawbacks. For example, routine examinations of material from field outbreaks of FMD in Great Britain showed that during the 1947-1951 epidemic, when 69 specimens were submitted, over a course of 56 months, only 58 percent could be directly identified after a single passage through cattle. In a second group of all samples received during November 1951 through June 1952 (8 months), 82 percent were typed by the complement-fixation test. The authors made no modification in the test, technique, or the sera used, but they believed that the higher percentage of samples successfully typed during the second period must have been due to the natural history of the disease at that time.

Results of the complement-fixation test to identify and differentiate the virus of VE from outbreaks in California, occurring from 1951 through 1955, indicated that the test can be used to a great advantage.[27,28] However, if the samples were not fresh and did not contain enough of the virus, the material had to be passed through hogs before the test could be successfully conducted. Although all samples submitted for testing did not fix complement, the difficulty may have been due to the fact that all types of the virus have not been recognized, and consequently the proper hyper-immune sera were not available.

Since VS virus fixes complement more readily than the other two vesicular viruses, the complement-fixation test has been adapted for the diagnosis and differentiation of this disease in the tests employed for the diagnosis of FMD and VE.[29,30,31]

The test described above is known as a direct complement-fixation technique. Because of idiosyncrasies of sera from individual cattle and swine, however, the indirect complement-fixation test may prove to be far superior. Dr. Rice of Canada and Dr. Brooksby of England have found this modification of the test to be a definite improvement in the diagnosis of FMD and VS.[32,33] Their work and results indicate that the indirect complement-fixation test may be far superior to the direct CF test presently employed in diagnosing VE.

Other laboratory methods, such as the virus neutralization test, can be used. In this test unknown serum containing antibodies is mixed with a known virus, and the mixture is inoculated into animals (or perhaps in tissue culture) to determine the presence or absence of antibodies.[8,20,34]

The cross-immunity test can be applied providing we have a number of animals known to be immune to the various diseases. Animals immune to FMD, VS, and VE can be inoculated with the suspected material. Susceptible animals that do not respond to inoculation, therefore, would constitute the identification of the causative agent. Such a test, however, would be complicated by the fact that we have at least

immunologically distinct types of FMD, six or more of VE, and two of VS; at least 14 animals recovered or hyper-immunized to the type specific diseases in question would, therefore, be needed.

The use of chicken embryonating eggs for the propagation of VS virus has simplified research and considerably increased our knowledge of this virus and the disease.[35]

Although many attempts were made, the virus of FMD was not successfully propagated in this manner until the work of Dr. Traub of Germany was uncovered soon after World War II. He had succeeded in cultivating FMD virus by mixing 10 strains of the virus and inoculating them into embryonating eggs. Although the strain of virus that actually was propagated was not known, Dr. Traub's work showed adaptation of the egg to propagation. His results could not be repeated until recently when the English workers confirmed his observations.

Thus far VE has not been propagated in chicken embryonating eggs; however, we have succeeded in growing five immunological types of the virus by grafting pieces of embryonic swine skin tissue on the chorio-allantoic membrane of chicken embryonating eggs. The embryonating eggs served only as a test tube and a medium. Although we have made many serial passages by this technique, we have been unable to adapt the strain of virus to grow in the chicken embryonating tissues.

VS quite regularly affects adult mice when injected intracerebrally, using either laboratory strains or on occasion field strains.[36,37] FMD, on the other hand, as a rule does not infect adult mice. FMD inoculated intramuscularly into mice six to eight days old (mice 9 days old or over show a resistance, and mice 6 days or younger are too difficult to handle) produces a dyspnea, a sluggishness, and later a muscular paralysis of the limbs and neck.[38] VE virus, on the other hand, has been found to be innocuous for either weaned or adult mice.[3,19] The differences in the infectivity of the three viruses for mice may serve as a laboratory means of differentiating between these three diseases.

Dr. Frankel of Holland[39] was the first to cultivate FMD virus in tissue cultures in sufficient quantities for vaccine production. The VE virus was propagated in a similar manner,[40] and VS can undoubtedly be grown very easily by this method. The monolayer technique, in which the cells are grown in a single layer on the bottom of a flask or tube and then inoculated with a virus, holds more promise.[41] This technique may be adopted to detect the virus in an unknown sample, and perhaps can be used in a neutralization technique by adding to the culture a specific anti-serum that would prohibit the growth of the virus, thereby serving to differentiate the virus being tested.

Transmission: Source of Infection.[6,7,10] The viruses of all three diseases -- FMD, VS, and VE -- are present in the fluid and the coverings of the vesicles, and they may be also found in the blood, in the organs, secretions, and excretions in the febrile stage of the infection. In the living animal the virus in the vesicles and other portions of the body tissues or organs, with rate exceptions, loses its infectivity in five to seven days after the appearance of lesions.

With FMD there is evidence that on some occasions the disease occurred in clean herds shortly after the addition of animals that had previously had the disease. In these cases, other sources of infection were eliminated for as long as eight months and in some instances for as long as a year after recovery. This would prove that some animals act as carriers of the infection; however, the percentage is believed to be very small. It has also been shown that the FMD virus may remain viable for some time on the skin, hair, loose portions, and crevasses in the hooves in the living animal.

Milk, meat, and raw byproducts of slaughtered and infected animals may also be instrumental in spreading the virus. Milk from febrile cases of FMD may contain the virus and when fed to susceptible animals may cause infection. There is evidence that the infectiousness of milk is lost during souring and fermentation that occur in cheese production.[6,42,43]

In the slaughtered animal the formation of lactic acid in the normal process of rigor mortis rapidly inactivates the FMD virus; however, quick-freezing suspends the acid formation and such muscle may remain infective for some time. Lymph nodes, liver, kidney, bone marrow, rumen, and other organs are not affected by the changes of rigor mortis and therefore may remain infective for some time depending upon circumstances.[7,34] A number of experiments have shown that FMD and VE may remain infective in meat that is properly refrigerated and frozen for at least six months.[42,44] A number of experiments have indicated that animals killed during the febrile stage of VE show the virus may live for one to several months if held at refrigeration temperatures (7° C) -- decomposition or gross putrefaction does not seem to have adverse effects on the virus. These results show the danger of slaughtering animals during the period of blood infectivity and the part such meat bone or meat scraps may play in the spread of the infection.

Most of the FMD outbreaks that have occurred in the United States were introduced through garbage originating in foreign countries.[42,45] The 1914 outbreak, the most serious and extensive ever known in this country (it spread into 22 states), had its origin in hogs fed trimmings and offal from a packing plant that handled foreign meats. This epizootic was not ended until 172,000 animals had been slaughtered, with a total expenditure of $4,600,000 by the Federal government and about $9,000,000 by the State governments.

We have found that hogs slaughtered 90 days after the last lesions of VE were healed and the herd released from quarantine could readily serve as a source of infection if garbage and scraps from such hogs were not cooked before being fed to susceptible swine. The tissues from hogs of this herd, consisting mostly of liver, spleen, and lymphatics, as well as bone marrow, were fed to a group of susceptible hogs, and infection resulted. It was not intended to prove that the animals were recovered carriers. The hogs may have been slaughtered during the stage of incubation before clinically visible lesions were observed, or they may have been suffering from a latent infection.[19]

Biological products manufactured on animals suffering from the disease can also disseminate an infection. In the 1908 outbreak vaccinia virus imported into the United States from Japan was contaminated with FMD virus.[46] The calves on which the smallpox virus was propagated recovered from the lesions and were later sold. The infection spread from the stockyards through the states of Michigan, New York, Pennsylvania, and Maryland. The disease was eradicated in about five months' time.

VS has been known for a number of years as a disease primarily of horses and sometimes cattle, with swine being highly susceptible to the virus when given experimentally by several routes of inoculation. The first natural outbreak of VS in swine confirmed by laboratory diagnosis was reported in 1943 in a Missouri hog cholera serum plant. This was found to be the New Jersey type. The source of this infection was never determined.

In the course of eradication of VE of swine, VS was encountered in several of the eastern states (Georgia, North Carolina, Virginia, Florida, Louisiana), indicating that the disease is far more widespread than heretofore recognized. The hogs on these ranches were fed grain and no known source of the virus could be found on the premises. The first and most important question concerned the origin of these infections. A committee was established to make an epizootiological study of the disease as a basis on which means of dealing with the problem could be recommended.

The disease was found to be enzootic, occurring year after year in this region on the same farm. The disease appeared to be seasonal, first observed in May or June, continuing through summer months, and usually last seen in September. Both cattle and swine were affected, and it appeared that the swine first developed the disease during the spring. Further studies indicated that the disease disappeared shortly after insect-killing frosts. The disease also was found to follow natural waterways, sparing farms away from water and infecting herds along the waterways. These data provide conditions characteristic of the habitat requirements of certain insects, such as stable flies, horse flies, black flies, and mosquitoes.

It was believed that the virus must survive the winter somehow, and so, to determine its source, a group of investigators from the Wisconsin Experiment Station was sent to the southeastern part of the United States to study the epizootiology and determine possible sources of the infection. Although this study is still in progress, a number of wild animals have been captured and their blood sera tested for the presence of antibodies. In all, 194 animals were taken during the summer months, including raccoons, foxes, bobcats, possum, wild hogs, squirrels, and other small animals and birds. Although the results of these tests are not yet complete, the raccoon was the first wild animal found to possess antibodies; 43 percent of the raccoons had sera that neutralized from 1,000 to 10,000 LD_{50} of the New Jersey VSV. The results of these tests, as well as the search for the virus in mosquitoes and earthworms, which are highly suspicious as disseminators of this disease, will be forthcoming. It is hoped that this investigation will discover means of transmission of this disease other than direct contact and that it will help to explain the persistence of the VS virus in certain regions of the United States and other countries.

Viability of the Viruses: When the vesicular viruses find themselves outside of the body, variable environmental conditions markedly influence their viability.[42] When thin films of FMD virus are exposed to sunlight it is readily destroyed, but when found in tissue fragments, such as in the epithelium of vesicles or on materials such as hair, feed, stable equipment, etc., the virus may remain infective for several weeks or more.

The virus of FMD is readily destroyed by heat. The usual pasteurization temperatures are fatal to it. At incubator temperatures the virus loses infectivity in 48 to 96 hours, depending upon the medium in which it is suspended. At room temperature it may remain viable somewhat longer, but at ordinary refrigeration (4° to 7° C) all three of the vesicular viruses may remain infective for many months, especially when placed in a buffered medium. When lyophilized or kept at temperatures of minus 40° to minus 70° C, the viruses remain viable for many years.[2,34,36,42,44]

When inoculated into sterile milk, the virus of FMD was found to remain viable for 30 days at refrigeration temperature, and it persisted much longer in cream than in skim milk. In butter made from sour cream the virus was rapidly destroyed. In salted butter made from sweet cream it remained viable for 14 days.

In sewage the virus may persist for some time, but if the sewage is enclosed, as in a septic tank, the fermenting process rapidly destroys it.

Although FMD virus is not considered to be unusually resistant when compared to other viral agents, its persistence under stable and farm conditions has been observed on several occasions. During the process of disease eradication as practiced in this country, restocking is not allowed for 30 to 60 days after the slaughter of affected and contact animals and a thorough cleaning and disinfection of the premises. Yet even under these circumstances in one instance in California the virus of FMD has been known to persist on the premises for as long as 345 days.

In the light of the rapid progress made with the new disinfectants some re-evaluation of their effect on vesicular viruses should be made; however, the old cold-tar disinfectants of the past have been rather disappointing. A detailed study of the effect of the generally recommended disinfectants upon FMD virus was made by Olitski, Traum, and Schoening.[34] They showed that alcohol in 70-75 percent solutions precipitated the protein fractions of the vesicular fluid and that the virus remained active for two to three days. It is apparent that the virus was surrounded by the coagulated protein and the infective agent was thus protected from direct contact with the alcohol. Ether, chloroform, phenol, bichloride of mercury, and cresol, were also found to be less effective against FMD virus than sodium hydroxide. A one percent solution of NaoH destroyed the virus in less than one minute. In practice sodium hydroxide at the rate of 2 percent has been found to be very effective and is used as a general disinfectant for all three of the vesicular viruses. Since this compound may prove to be corrosive and dangerous to people not acquainted with its action a 4 to 5 percent sodium carbonate solution may be used, if sufficient time is allowed for its action.

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FOOT-AND-MOUTH DISEASE IN MEXICO

Mexican-United States Commission for the Prevention of Foot-and-Mouth Disease

The history of animal disease eradication is rather marked by the absence of concerted action between nations. In 1947 Mexico and the United States set a precedent when they combined their forces in an all-out effort to eliminate foot-and-mouth disease from Mexico. Both Nations can be justly proud of the cooperative spirit that has prevailed throughout this campaign.

The dreaded words "Foot-and-Mouth Disease" first appeared in Mexican newspapers in the latter part of 1946. The first outbreak occurred near the City of Veracruz and the disease was first officially diagnosed in the State of Puebla on December 26, 1946. By the time a joint U.S.-Mexico Eradication Commission could be formally established in April 1947, the disease had swept like wild fire from the Gulf of Mexico to the Pacific Ocean involving 16 states and the Federal District. This comprises about one-fourth of Mexico's land area. As rapidly as possible, quarantine lines were set up around the infected zone. Inside were some 17,000,000 animals infected with or exposed to the disease. Through a five point program of inspection, vaccination, quarantine, disinfection, and eradication, the disease was brought under control and vaccination activities were terminated in August 1950 and emphasis was placed on inspection and vigilance.

A one-year period elapsed before a small outbreak occurred in the State of Veracruz. It was quickly eradicated, but seven months later, the disease again appeared in the State of Veracruz and was again promptly eradicated.

21 months later, the third and last outbreak appeared near the City of Gutierrez Zamora, Ver., on May 23, 1953. So far as can be determined there was no relationship between these three outbreaks except that they occurred in the same general vicinity.

At the inception of the Gutierrez Zamora outbreak, the small vigilance force still working in Mexico was quickly dispatched to the scene of the outbreak to begin operations.

As might be expected the organization experienced many difficulties in our efforts to stamp out the disease. The focus of original infection consisted of 160 square miles of rough tropical terrain. For many months daily animal inspections were begun in and around the infected zone on horseback, by boat, and by vehicle.

In order to maintain a rigid quarantine on the 45-mile perimeter of the original infected zone, a total of 402 Mexican soldiers was employed. A secondary quarantine line 120 miles in length was patrolled by 94 soldiers. An additional 126 soldiers were utilized at the 81 quarantine and disinfection stations located at strategic points in and around the zone of operations.

In order to check on the possibility of the disease having extended itself outside the quarantined zone, two teams of livestock inspectors were selected to inspect and make inquiries at stockyards and slaughter houses in the triangular zone between Tampico, Mexico City and the Port of Veracruz. Animal movements in this triangular zone were under close surveillance. For the purpose of alerting Mexican Government Regional veterinarians and livestock owners, a team of veterinarians traveled throughout the rather extensive zone that had been under quarantine in 1947-52.

The Commission found it necessary to increase its personnel to a force that included in 1954 more than 2,300 employees.

By the time the disease was brought under control in April 1954, 41 sporadic outbreaks had occurred but none more than 31 miles from the original focus of infection.

Following the elimination of the disease, 20,000 premises were cleaned and disinfected. In order to test the efficiency of cleaning and disinfection procedures, 2,600 test animals were introduced on to the formerly infected premises and subjected to a 90-day exposure period. Vigilance committees composed of Mexican livestock owners and cattlemen were organized throughout the zone of operations and adjacent regions and instructed to report any suspicious animals to the Commission immediately.

At the present time our organization includes 3 American veterinarians and one Administrative Officer, a like number of Mexican counterparts and 14 Joint Commission employees. Central offices are located in Mexico City. A shop and warehouse are functioning to care for vehicles and emergency equipment that have been retained for use should an outbreak occur. A diagnostic laboratory has been retained for our use in the event it becomes necessary to run serological or biological tests.

In addition to detecting and investigating conditions of a suspicious nature, present personnel make periodic field trips to observe animals throughout Mexico and to renew contacts with livestock owners and former employees.

We are ready to begin within minutes, day or night, operations to stamp out any foot-and-mouth disease that might appear in Mexico.

As of this date, however, there have been no outbreaks of foot-and-mouth disease reported in Mexico for more than 22 months. The last three outbreaks have occurred in a relatively small area in the State of Veracruz. The remainder of Mexico has, therefore, been free of the infection for more than six years.

It is perhaps unnecessary to mention here the unpredictable characteristics of foot-and-mouth disease. It is because of these characteristics, however, that vigilance continues even though it appears eradication has been achieved.

We wish to emphasize that the favorable conditions existing in Mexico are the result of the cooperation of the Mexican people and their officials; Secretary of Agriculture, Gilberto Flores Munoz; Sub-secretary for Livestock, Dr. Lauro Ortega, and Commission Technical Director, Dr. Gabriel Fernandez de Castro.

RIFT VALLEY FEVER

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Introduction: There are many exotic diseases which probably would never spread even if infected animals were introduced into this country. I am thinking particularly of those diseases which are dependent on special insect vectors for transmission, e.g., trypanosomiasis (tsetse flies), leishmaniasis (Phlebotomus spp), East Coast fever (ticks of the Rhipicephalus spp), and heartwater (bont-ticks), etc. These diseases, however, in the presence of their specific vector, are a different proposition and are the major disease problems of Africa, making diseases like brucellosis and tuberculosis appear almost unimportant by comparison. And, while this is generally so, one cannot be too sure when one thinks how anaplasmosis is behaving in the U. S. A. This disease in Africa is regarded as entirely dependent on tick transmission since it does not occur in tick-free areas. As you know, this disease is becoming an increasing problem in this country, and this in the absence of ticks!

But there are a number of other exotic diseases which are a great menace since they could spread like wildfire in this country. Most of these are of the highly contagious type like African swine fever, fowl plague, rinderpest, foot-and-mouth disease, and other similar diseases which do not need the presence of special insects for transmission, but there are others which even though dependent on insect transmission would probably spread here. I am thinking of diseases like yellow fever, rabbit myxomatosis, and Rift Valley fever, the vectors of which are either already present or which could probably be spread by other insects present in the U. S. A.

Definition: Rift Valley fever is an acute infectious disease of sheep, cattle and other animals, including man. Its importance to the animal industry is the high abortion rate produced in pregnant cattle and sheep and the high mortality rate in lambs and calves. The disease is caused by a small virus which is transmitted to animals by a variety of mosquitoes and other insects but is often contracted by humans due to contact, as during the performance of an autopsy.

Etiology: The causative organism is a small virus (±30mu) which is closely related to that of yellow fever. It can easily be adapted to embryonated eggs. This virus has a particularly wide host range, being much wider than most viruses.

History: Like many other diseases which are called after place names, this is another. It is named after the Rift Valley of East Africa.

It has been encountered in several outbreaks in Kenya, beginning in 1913. In these outbreaks, heavy mortality was experienced in young lambs but it was only in 1931 that Daubney, Hudson and Garnham isolated and described the virus and they also recognized its infectivity for the human when some 200 cases were reported in humans. Although neutralizing antibodies have been found in sera of humans in a number of other Central African localities, the virus itself has also been encountered in Uganda in 1944-47 by a team of Rockefeller Institute workers led by Smithburn. I would like to point out that in these Central African areas there are only small populations of cattle and sheep and this is probably why the disease has not often been noticed in these areas in an active form.

Up to 1951, the only cases of the disease were the odd reports from Central Africa and the occasional laboratory infections in England, the United States, and Japan.

Apart from the rather limited outbreaks in Kenya, the diseases was not regarded of economic importance until in 1951 when it appeared in a sudden widespread outbreak in South Africa, in which it is estimated that it caused the loss of 100,000 sheep and some 20,000 human infections. In this outbreak, losses were being suffered in sheep but it was not realized at first that they were due to a new disease and were variously ascribed to bluetongue, enterotoxemia, vegetable poisoning, internal parasites, and other infections. The human cases were thought to be influenza. It was only after the disease had spread considerably that a pedigree bull died suddenly in rather a surprising way. This case created the curiosity of five veterinarians who autopsied it, but within the next five days three of them became ill. This aroused great suspicion and an immediate investigation was made as to the animal virus diseases that could cause similar human infection. We could think of only two, namely Rift Valley fever and Q-fever. Today, we would have to think of at least one more - the Wesselsbron virus which has been the cause of abortions in sheep in South Africa. Then there was a search for antisera against these diseases. No Rift Valley fever antiserum was available in South Africa, but we were fortunate in obtaining a supply from Nairobi, thousands of miles away. From then on it did not take long to confirm the diagnosis of Rift Valley fever, but it was still a long and big task to determine the extent of spread and the development of a suitable vaccine.

I have deliberately mentioned these incidences because we in this country should profit from these experiences and not be caught in the same situation. We should anticipate these diseases and we should be prepared in advance to meet such an emergency. Every suspicious and unusual outbreak of disease should be thoroughly investigated in the light of these exotic diseases.

Epizootiology: The distribution has already been mentioned.

In Kenya the disease has the habit of appearing periodically in flocks in more or less explosive outbreaks, at intervals of 5-7 years. It seems that in nature this is a jungle disease living a cycle between insects and rodents and its appearance in domestication is more or less an accident, but when it appears, and if the right conditions exist - namely, a large population of susceptible animals and large numbers of vectors, an epizootic of a most explosive type is set off. During such an outbreak the disease spread with remarkable speed and usually within a few weeks or months the epizootic will have taken its course and disappeared, but not before widespread infection has occurred. The morbidity rate is very high.

The carriers of the disease are still being investigated but so far seven species of mosquitoes and one other insect have definitely been associated with transmission, and it is highly probable that during an outbreak many kinds of blood sucking insects would act as mechanical vectors. I feel that if this virus were introduced into this country in the summer or fall that widespread outbreaks could occur. I would also like to note that the type of country in which the extensive outbreaks occurred in South Africa resembles the prairieland of this country very closely.

Symptoms: There are no pathognomonic clinical features.

The chief characteristics of this disease are an abrupt onset, short course, a sharp febrile reaction, high death rate in lambs and calves, but much lower in adult sheep (less 20 percent) and adult cattle (less 10 percent), but probably its most characteristic feature is a very high abortion rate in pregnant cattle and sheep. Abortions occur at all stages of pregnancy.

In cattle and sheep death is sudden, almost without warning, but if one were able to record the rectal temperature one would have noticed a high fever for a day or two prior to death.

In its typical form the disease has a brief and abrupt course. Chronic and lingering cases are rare in all animals.

In the human the incubation period is slightly longer being on an average 4-5 days. Often the disease is mistaken for influenza. This is due to its brief course and a similar febrile reaction with headaches, but an important difference is that the catarrh of the upper respiratory tract is absent in Rift Valley fever. An occasional but important eye lesion is the hemorrhage into the retina causing temporary or permanent impairment of vision.

Mortality Rate: In young calves and lambs it can be as high as 90 percent, and in adult sheep less than 20 percent, and adult cattle less than 10 percent. In mice the death rate by artificial infection is about 99 percent. In the human it is generally considered as non-fatal though two cases have been ascribed to it. This is out of thousands of cases.

Communicability: In animals the disease usually does not transmit in the absence of insect vectors. For instance, one can have infected and susceptible sheep or mice together without danger of transmission to one another, but humans easily become infected if they handle any material rich in virus, like infected blood, while performing an autopsy.

Pathological Changes: The most significant and consistent lesions are in the liver.

Macroscopically these in their most typical form consist of small circumscribed greyish or dark foci disseminated throughout the substance of the liver. In several cases where these lesions have coalesced the entire liver is necrotic and sometimes the liver is yellow or light brown due to icterus. Ordinarily the liver is not enlarged.

The other lesions are less typical and are found in many acute febrile diseases - general venous congestion, petechial hemorrhages on the heart, in lymph glands and in the alimentary tract. The spleen may be slightly enlarged but often is quite normal.

Microscopically: The principal lesions are seen in the liver and are almost pathognomonic.

The lesion is essentially a circumscribed focal necrosis. Apparently, as the inflammatory reaction develops, histiocytes and neutrophiles which invade the lesion also become degenerated so that the entire lesion consists of degenerated liver cells and leucocytes. The size of the foci depends on the severity of the disease - in the most acute cases they are very extensive, having coalesced.

Acidophilic nuclear inclusions are also considered to be characteristic of Rift Valley fever. They are circumscribed and vary in size and number. These inclusions are most frequent in the livers of mice or young lambs and less so in the livers of adult sheep and cattle, particularly if these were sacrificed and did not die a natural death.

Diagnosis: The circumstantial evidence is usually indicative of the disease - namely:

(a) human infections with short febrile course.

(b) high mortality rates in lambs and calves, but lower mortality rates in adult sheep and cattle.

(c) high abortion rates in pregnant cattle and sheep.

(d) typical liver lesions in autopsied animals.

The definite diagnosis depends upon:

(a) virus isolation in the active disease.

White Swiss mice are extremely susceptible and consistently die within 2-4 days, after i.p. injection. This is the only virus that will cause death in mice as quickly as this though bacteria like anthrax and some clostridia may also do this, but if antibiotics had been added to the inoculum these would be eliminated anyway. A specific diagnosis can be made directly by dividing the inoculum into two parts and the respective portions treated with negative and positive sera. Results are very clear-cut and as a rule a diagnosis can be made within four days. The livers of dying mice are collected in 10 percent formalin for histological examination.

The simultaneous inoculation of sheep with the suspected material will also reveal a rather typical response. The typical reaction has already been described.

Serologically: In convalescent or recovered cases it is an easy matter to demonstrate the presence of antibody since this is present in abundance and the two tests usually used give clear-cut results, namely the serum neutralization and complement-fixation tests. The antibody becomes demonstrable within a week after infection and persists for a long time - in the human it is known that after twenty years these are still very powerful.

Differential Diagnosis: This would be concerned with three or four important features of the disease.

(a) Human infections with a short febrile course.

(1) Dengue and phlebotomus fever have a rather similar febrile reaction.

(2) Q-fever.

(3) Influenza.

(4) Yellow fever.

(5) Wesselsbron virus.

(b) High mortality rate in lambs and calves and a lower mortality in adult animals.

    (1) Enterotoxemia.
    (2) Vegetable poisoning.
    (3) Bluetongue.
    (4) Acute bacterial infections.
    (5) Helminthiasis.
    (6) Nairobi sheep disease.
    (7) Heartwater.
    (8) Wesselsbron virus.

(c) Abortion.

    (1) Sheep

        (a) Nairobi sheep disease.
        (b) Ovine enzootic abortion.
        (c) Vibriosis.
        (d) Wesselsbron virus.

    (2) Cattle

        (a) Brucellosis.
        (b) Trichomoniasis.
        (c) Vibriosis.
        (d) Leptospirosis.

NEWCASTLE DISEASE (EXOTIC FORM)

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Newcastle disease is recognized in two different forms:

1. Exotic form (European and Asiatic form) characterized by high mortality (75 percent or greater), rapid spread, and by necrotic and hemorrhagic lesions in the proventriculus and intestinal tract.

2. American form characterized by lower, but variable, mortality, respiratory and nervous manifestations, and usually by absence of hemorrhages and necrosis in the intestinal tract.

The fact that the disease occurred in this latter form made it unrecognized in the early 1940's when it became a disease problem in California. It is probably also the reason why we have Newcastle disease in this country at the present time because had it occurred in its original exotic form, it would have been recognized and promptly eradicated.

Historically, the virulent form of Newcastle disease was the one that was first recognized as a disease entity in the Dutch East Indies in 1926 by Kraneveld. Doyle in England studied the disease about the same time. He isolated the causative agent and gave the disease its name - Newcastle disease - after the locality of the first outbreak near Newcastle-on-the-Tyne in England.

Etiology: A filtrable virus, about 100 mu in size, is the cause of Newcastle disease. Both the exotic and American forms of the disease are caused by strains of virus which are immunologically similar.

A few of the chief properties of the virus might be mentioned:

1. The virus grows readily in the developing chicken embryo where it causes death of the embryo in 48 to 72 hours. Dead embryos are markedly congested and skin hemorrhages may be prominent. Yolk sac congestion and hemorrhages are also observed.

2. Virus-infected fluid from inoculated eggs has the property of clumping washed red blood cells of the chicken and certain other species. This phenomenon

is known as hemagglutination and is the basis of the hemagglutination-inhibition (HI) test which is a laboratory test for detection of serum antibodies.

3. The virus of Newcastle disease is variable. Different strains vary greatly in pathogenicity. For example, the exotic strains are the most pathogenic with enterotropic properties while the American strains are less pathogenic with pneumotropic and neurotropic tendencies. Even among the American strains there is considerable variation in pathogenicity. The B_1 intranasal vaccine strain is one of the least pathogenic, being incapable of infecting day-old chicks when inoculated intracerebrally, while most all other strains are lethal for chicks when inoculated by this route. In contrast to the B_1 strain is the GB-Texas-1948 American strain which can cause mortalities of over 50 percent in adult laying flocks. Death follows complete paralysis.

Hosts: While domestic fowl are the usual hosts a number of species of birds have been found susceptible to the virus. This fact is important from the standpoint of control because free flying birds might be potential spreaders from infected areas.

Clinical Manifestations of the Exotic Form: The onset is sudden, the chickens become depressed, go off feed, and diarrhea occurs early. Later the droppings become scant and consist primarily of urates and bile. Many of the birds have difficult breathing with a prolonged inspiratory act; some tracheal rales may be heard. There may be a collection of mucus in the mouth and nostrils and cyanosis of the head may be observed. There is progressive weakness followed by complete prostration and death in 24 to 48 hours. In prolonged cases, nervous manifestations may be observed. In pigeons, ducks and geese paralytic symptoms are predominant.

Pathology: At necropsy the most outstanding lesions are found in the intestinal tract associated with the lymphoid follicles. These areas may be hemorrhagic and necrotic. The lesions may be seen from the serosal surface as swollen, congested or hemorrhagic areas. The mucosal surface of the proventriculus may also be markedly congested or even hemorrhagic. The spleen may show greyish areas of focal necrosis. The breast muscle may have a streaked appearance because of degenerative changes. Corneal opacity is sometimes observed.

Diagnosis: A tentative field diagnosis of the exotic form of Newcastle disease may be made from the clinical manifestations, mortality, and necropsy findings. Since fowl plague also causes similar gross lesions,

it is necessary to have the diagnosis confirmed by isolation and identification of the virus. Serological studies may aid in diagnosis, but could not be used to differentiate the exotic form from the American form of the disease.

In differential diagnosis, consideration must be given to the American form and to fowl plague. To differentiate the exotic strains from the American strains, laboratory tests must be made to determine the pathogenicity of the strain involved. The incidence of hemorrhagic and necrotic lesions in the intestine is much greater for the exotic strains than for the American strains. Also, clinical manifestations help differentiate the strains.

It may be difficult to differentiate fowl plague and the exotic form of Newcastle disease on the basis of clinical signs and necropsy findings. In the laboratory differentiation can be based on the use of known positive serum. Inoculation of mice with fowl plague virus causes infection while pigeons are generally resistant. With Newcastle disease virus toxic effects may be noted in mice and pigeons are susceptible. Differences are also observed in the hemagglutination of horse erythrocytes.

Control: A suspected flock should be quarantined until a definite diagnosis can be established. The fact that the exotic form of Newcastle disease can usually be recognized readily makes possible eradication by quarantine and slaughter methods. The success of this method has been proved on several occasions in England and Australia, and the 1950 outbreak in California resulting from the importation of game birds. Import restrictions are aimed at foreign diseases such as this one. These restrictions are:

1. A permit before leaving the country of origin.

2. A veterinary inspection of the flock and premises must demonstrate freedom from fowl plague or Newcastle disease.

3. Veterinary inspection at the port of entry.

4. Quarantine a minimum of 15 days at the port of entry.

NEWCASTLE DISEASE AND FOWL PLAGUE

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The terminology used by various writers in several countries to describe Newcastle disease and fowl plague is confusing. Fowl pest has been used synonymously for both of these diseases. To prevent further confusion, it is suggested the term "fowl pest" be discarded.

Newcastle disease, caused by a virus, is characterized by respiratory and nervous symptoms. It was first described in 1926 in Java, and in 1927 in the area of Newcastle, England, from which the name Newcastle disease was derived.

In 1940, in the Petaluma area of California, a peculiar disease of chickens was studied. There were respiratory symptoms, followed by or accompanied with nervous symptoms. It was called respiratory-nervous disorder, and later given the descriptive name of pneumoencephalitis. The cause was found to be a virus, but it was not until 1945 when the disease spread to other parts of the United States, that serological tests proved it was a type of Newcastle disease of a mild nature, compared with the European outbreaks.

The respiratory phase is characterized by gasping symptoms, not unlike infectious bronchitis. In chicks and growing stock, a nervous phase later develops, with a twisting of the neck to one side, or downward and backward, or the head may extend upward and backward. Affected individuals may fall to the side, or sit and suffer clonic spasms.

Laying hens may or may not show respiratory distress. Egg quality is affected, with rough or thin shells, and liquid albumin. Production lessens and some birds will stop laying. In severe cases nervous symptoms are prominent. The highly fatal type affects birds of all ages, and the mortality is high.

Lesions on postmortem examination are not striking, except in the highly fatal form. Of most significance is an enlargement of the spleen and a cloudiness of the air sac tissues. The air sacs may be slightly thickened and grey in color. Mucus may be observed as bubbles on the air sacs, and a slimy exudate is noticed, particularly to the touch. The fatal form produces hemorrhages in the mucosa of the proventriculus and in the intestines, particularly the caeca and rectum.

Fowl plague, a highly fatal disease, was first observed in Italy in 1878, but soon spread to other European countries. It was not until

1900 that the cause was found to be a virus. Most of the avian species are affected, but water fowl and pigeons are less susceptible.

The symptoms are acute and appear to be of a septicemic nature, much like fowl cholera. There is a high temperature, depression, and inappetance. The eyelids, ear lobes, comb and wattles are edematous and have a pale blue-like color. A thick, sticky mucus develops in the eyes, nostrils, and mouth. As a result, there may be difficult breathing. Coma and convulsions are common terminal symptoms.

Postmortem lesions are pronounced hemorrhages of the musculature, which may be observed on cross section. Petechiae and ecchymoses are present on the pleura, heart, liver, spleen, abdominal fat, and in the proventriculus, gizzard, and lower intestinal mucosa.

The differential diagnosis of Newcastle disease and fowl plague creates some problems. Both viruses will agglutinate chicken red blood cells, and they are similar in some other respects. The comparatively mild type of Newcastle disease usually seen in the United States is not difficult to diagnose. However, it must be remembered that because of its mildness it was not properly identified for perhaps ten years after its first appearance. Fowl plague is reported to occur in Egypt in a much less severe form, and it could be easily confused with some of the more common local diseases of poultry.

Newcastle disease seems to spread more rapidly than fowl plague. Pigeons and water fowl are less susceptible to the latter. The respiratory and then the nervous symptoms of Newcastle disease are characteristic. No symptoms may be seen with fowl plague, but if there is respiratory distress, it is the result of an accumulation of thick mucus in the mouth, and perhaps nostrils. Also, the nervous symptoms of coma and convulsions are those occurring at death. Of most significance is plaques on the comb, which may be followed by necrosis and a severe edema and cyanosis of the face, comb, and wattles.

Postmortem lesions are usually mild with Newcastle disease, however, the highly fatal type produces hemorrhages of the proventriculus, caeca and rectum. Hemorrhages of the muscles throughout the abdominal organs, including the mucosa of the proventriculus, gizzard, and lower intestines, are striking lesions of fowl plague.

Definite differential diagnosis is made by egg embryo and animal inoculation, histopathology, and serological tests. While both viruses will agglutinate red blood chicken cells, fowl plague will also agglutinate horse and sheep cells. Cross immunity and serum neutralization tests are confirmatory.

The fatal type of Newcastle disease and fowl plague have similar characteristics. A mild type of fowl plague might occur which could be easily confused with Newcastle disease, as it usually occurs in the United States, as well as some of the more common diseases, such as fowl cholera, and some of the complex respiratory diseases.

COMPARISON EXOTIC NEWCASTLE DISEASE AND FOWL PLAGUE

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CHARACTERISTIC	EXOTIC NEWCASTLE	FOWL PLAGUE
First recognized	1926 by Doyle	1878 in Italy by Perroncito
Synonyms	Pneumoencephalitis Pseudo fowl pest	Fowl pest
Distribution	Orient, Europe, U.S.A. by imports, S. America	Orient, Europe, South America, U.S.A., 1924-25, 1929
Etiology	Virus	Virus
Definition	Acute highly fatal rapid spreading disease	Acute highly fatal disease
Duration	Death 3-4 days	Death in 2 days
Symtoms:	Acute viremia	Acute viremia
Cyanosis	Characteristic	Marked
Conjunctiva	Less often observed	Red and swollen
Edema of head and neck	Less often observed	Typical
Edema of glottus	Not common	Typical
Exudate from nostrils	Common	Typical
Diarrhea	Common	Common
Nervous	Frequently noted	Observed in lingering cases
General malaise	Observed	Head resting on floor, typical
Focal areas of necrosis comb and wattles	Not observed	On comb and wattles
Birds affected	Many	Turkey, chicken, most birds resistant
Laboratory animals	Mice, hamsters	Refractory
Man	Conjunctivitis	Refractory
Respiratory	Typical	Frequently observed
Rate of spread	Very rapid	Very rapid
Mode of infection	Respiratory & digestive	Respiratory & digestive
Contact exposure	Readily transmitted	May or may not be transmitted
Pathology:	Septicemia	Septicemia
Petechia	Internal organs, fat muscles	Internal organs, fat heart. Proventriculus, body muscles under gizzard lining
Ecchymoses	Common	Common

CHARACTERISTIC	EXOTIC NEWCASTLE	FOWL PLAGUE
Pathology (Cont'd)		
Rigor mortis	Not so quickly (as pest)	Develops very soon after death
Edema head	Not commonly observed	Typical straw-colored clear fluids
Liver	Not characteristic	Not characteristic
Lungs	Congestion, edema, fibrinous casts	Not characteristic
Intestines	Petechial hemorrhages of mucosa	Petechial hemorrhages of mucosa, no hemorrhagic enteritis
Ovary	Engorged	Highly engorged
Oviduct	Not characteristic	Gray exudate
Microscopic foci	Various tissues	Necrosis various organs
Nature of Virus:		
Hemagglutination	Hemagglutinates red cells of various animals	Hemagglutinates red cells of various animals
Exposure to sun	Quickly destroyed	Quickly destroyed
Exposure to chemicals	Fairly easily destroyed	Easily killed by disinfectants
Refrigeration	Resistant for years	Resistant for years
pH effects	Most resistant	Destroyed pH 4
Local of virus	Most body tissues	All body tissues
Antibiotics	Resistant	Resistant
Room temperatures	Fairly resistant	Fairly resistant
HI	Easily demonstrated	Easily demonstrated
HI	Specific for NCD	Specific for fowl plague
Carriers	Possibly?	Possibly recovered individuals
Disease Most Confused	Infectious bronchitis (CRD)	Fowl cholera
Other diseases	Respiratory &nervous infections	Botulism, poisoning
Diagnosis	Chicken embryo isolation of virus	Chicken embryo isolation of virus
HI Test	Specific known antisera	Specific known antisera
Virus neutralization	Specific antisera	Specific antisera
Bacterial	Negative	Negative
Susceptible Chickens	3-5 days	36-72 hours
Control	Burn, destroy, disinfect immune zone barrier	Burn, destroy, disinfect vaccines unsatisfactory

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